********** STN Columbus **********

FILE 'HOME' ENTERED AT 12:58:40 ON 06 MAR 2001

=> file biosis caba caplus embase lifesci medline scisearch uspatfull japio
=> e mardh sven/au

E1 1 MARDH PERANDERS/AU
E2 337 MARDH S/AU
E3 39 --> MARDH SVEN/AU
E4 6 MARDHEKAR B V/AU
E5 1 MARDHEKAR DHANANJAY V/AU
E6 2 MARDHIAH MOHD ZIN/AU
E7 10 MARDHY A/AU
E8 4 MARDHY ABDELHAKIM/AU
E9 7 MARDI A/AU
E10 1 MARDI A R/AU
E11 1 MARDI A S/AU
E12 1 MARDI AGUS/AU

=> s e2-e3 and (gastri? or pylori)

L1 243 ("MARDH S"/AU OR "MARDH SVEN"/AU) AND (GASTRI? OR PYLORI)

=> dup rem l1

PROCESSING COMPLETED FOR L1
L2 86 DUP REM L1 (157 DUPLICATES REMOVED)

=> d bib ab 1-

YOU HAVE REQUESTED DATA FROM 86 ANSWERS - CONTINUE? Y/(N):y

L2 ANSWER 1 OF 86 CAPLUS COPYRIGHT 2001 ACS
AN 2001:128060 CAPLUS
TI Characterization of oxyntic glands isolated from the rat ***gastric*** mucosa
AU Azerkan, L.; Bengtsson, P.; Tommeras, K.; Li, Z.-Q.; ***Mardh, S.***
CS Faculty of Health Sciences, Division of Cell Biology, Department of Biomedicine and Surgery, Linkoping University, S-581 85, Linkoping, Swed.
SO Comp. Biochem. Physiol., Part A: Mol. Integr. Physiol. (2001), 128(2), 349-357
CODEN: CBPAB5; ISSN: 1095-6433
PB Elsevier Science Inc.
DT Journal
LA English
AB A simple and reproducible method for isolating oxyntic glands from the rat ***gastric*** mucosa was developed. The mucosa was incubated with pronase and EGTA, and then treated mech. to release glands that were sepd. from single cells by sedimentation. Parietal cells were identified by immunostaining using a monoclonal antibody against H,K-ATPase. The glandular cells appeared morphol. intact. By careful control of the conditions of gland isolation, long glandular structures comprising hundreds of cells surrounding the lumen were obtained. I.p. injection of

Br-deoxyuridine in the rat 1.5 h before the isolation procedure resulted in glands with a labeling of cells in their neck region. The glands were viable, as demonstrated by their ability to respond to various hormones. Histamine dose-dependently stimulated the acid formation which was measured as the accumulation of [14C]aminopyrine. At 100 .mu.M histamine the accumulation was increased 5-10-fold. At 100 nM, pentagastrin potentiated the histamine stimulated accumulation by approx. 40% but pentagastrin alone did not stimulate. The oxyntic glands obtained by the present procedure appear useful for studies on cell physiol., including regulation of acid secretion, cellular interactions, and possibly also differentiation and proliferation mechanisms since long glandular fragments that contained the proliferative zone could be isolated.

L2 ANSWER 2 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 1
AN 2001:25050 BIOSIS
DN PREV200100025050
TI Omeprazole and CYP2C19 polymorphism: Effects of long-term treatment on ***gastrin*** , pepsinogen I, and chromogranin A in patients with acid related disorders.
AU Sagar, M.; Bertilsson, L.; Stridsberg, M.; Kjellin, A.; ***Mardh, S.*** ; Seensalu, R. (1)
CS (1) Department of Medicine, St Gorans Sjukhus AB, S-112 81, Stockholm: Rein.Seensalu@stgoran.se Sweden
SO Alimentary Pharmacology & Therapeutics, (November, 2000) Vol. 14, No. 11, pp. 1495-1502. print.
ISSN: 0269-2813.
DT Article
LA English
SL English
AB Background: The polymorphic enzyme CYP2C19 is of importance for the metabolism and effects of omeprazole during short-term treatment. Aim: To investigate the relationship between CYP2C19 genotype and the effects of long-term omeprazole treatment. Material and methods: A total of 180 patients with acid related disorders were genotyped for wild type and mutated CYP2C19 alleles by allele-specific PCR amplification. ***Gastrin*** and chromogranin A were assessed by radioimmunoassays, and pepsinogen I and H. ***pylori*** serology were assessed by ELISA methods. Results: In 108 of the patients, who received a single dose of 20 mg omeprazole, there was no difference in ***gastrin*** and chromogranin A concentrations between the three CYP2C19 genotypes. In 72 patients on long-term treatment (> 1 year) with 20 mg omeprazole daily, serum ***gastrin*** as well as plasma chromogranin A concentrations (mean +- s.e.) were both about threefold higher in the wild type/mutated (52.1 +- 7.6 pM and 7.3 +- 1.3 nM (n = 19), respectively) compared to wild type/wild type (14.7 +- 0.9 pM and 2.5 +- 0.1 nM (n = 52), respectively; both comparisons P = 0.0001). In a single mutated/mutated patient on long-term treatment, both ***gastrin*** and chromogranin A were high (88 pM and 13.7 nM, respectively). Serum pepsinogen I concentration was significantly lower in wild type/mutated (n = 19) patients on long-term treatment, compared with the corresponding wild type/wild type (n = 49) group (147 +- 19 mug/L vs. 193 +- 12 mug/L, P = 0.04). Conclusion: Patients with one (and probably also with two) mutated CYP2C19 allele(s) on long-term treatment with omeprazole had significantly affected serum ***gastrin*** and pepsinogen I and plasma chromogranin A concentrations compared with patients with two normal alleles. This indicates that

changes in ***gastric*** mucosal morphology during omeprazole treatment might be dependent upon the degree of the individual's capacity to metabolize omeprazole.

L2 ANSWER 3 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 2
AN 2000:406170 BIOSIS
DN PREV200000406170
TI Prevalence of gastroduodenitis and Helicobacter ***pylori*** infection in a general population sample: Relations to symptomatology and life-style.
AU Borch, Kurt (1); Jonsson, Kjell-Ake; Petersson, Fredrik; Redeen, Stefan; ***Mardh, Sven*** ; Franzen, Lennart E.
CS (1) Department of Surgery, University Hospital, S-58185, Linkoping Sweden
SO Digestive Diseases and Sciences, (July, 2000) Vol. 45, No. 7, pp. 1322-1329. print.
ISSN: 0163-2116.
DT Article
LA English
SL English
AB Some benign and malignant diseases develop on the background of chronic ***gastritis*** or duodenitis. The present study was performed in order to determine the magnitude of these background changes with relations to symptomatology and life style in the general population. Examinations were performed in 501 volunteers (age 35-85 years). Fifty percent had ***gastritis*** ; this was associated with H. ***pylori*** in 87%. H. ***pylori*** -negative ***gastritis*** was associated with regular use of NSAIDs (odds ratio 3.8 (1.6-9.9)). Duodenitis, observed in 32%, was associated with H. ***pylori*** infection (odds ratio 2.3 (1.3-4.6)), previous cholecystectomy (odds ratio 3.6 (1.1-16.1)), and regular use of NSAIDs (odds ratio 3.0 (1.4-7.1)). Neither ***gastritis*** nor duodenitis was associated with smoking or alcohol consumption. The rate of digestive symptoms did not differ between subjects with and without uncomplicated ***gastritis*** or duodenitis. In conclusion, half of this adult population had ***gastritis*** strongly associated with H. ***pylori*** infection. ***Gastritis*** without H. ***pylori*** infection was frequently associated with regular NSAID intake. One third had duodenitis, which was associated with H. ***pylori*** infection as well as with regular use of NSAIDs and previous cholecystectomy. Digestive symptoms were not overrepresented in uncomplicated ***gastritis*** or duodenitis.

L2 ANSWER 4 OF 86 CAPLUS COPYRIGHT 2001 ACS
AN 2000:788838 CAPLUS
DN 134:143721
TI N-terminal phosphorylation of ***gastric*** H/K-ATPases both in vitro and in vivo
AU Kanagawa, M.; Umezu, H.; Kaya, S.; Watanabe, S.; Kagawa, I.; Shimada, A.; Imagawa, T.; ***Mardh, S.*** ; Taniguchi, K.
CS Biological Chemistry, Division of Chemistry, Graduated School of Science, Hokkaido University, Sapporo, 060-0852, Japan
SO Int. Congr. Ser. (2000), 1207(Na/K-ATPase and Related ATPases), 587-590
CODEN: EXMDA4; ISSN: 0531-5131
PB Elsevier Science B.V.
DT Journal
LA English

AB Acid secretion is regulated by second messenger pathways. Histamine stimulates acid secretion via the H2 receptor-mediated activation of cAMP dependent protein kinase. Acetylcholine also stimulates acid secretion by increasing protein kinase C activity, via an increase in intercellular calcium concns. This protein kinase-dependent acid secretion is regarded to be coupled with intercellular protein phosphorylation. Tyr kinase modifiers regulate acid secretion. The Tyr-10 and Tyr-7 in the .alpha.-chain of not only pig but also rat and rabbit stomach H/K-ATPase of the G1 fraction are reversibly phosphorylated by endogenous Tyr kinase and phosphatase. Time course of phosphotyrosine (PY) formation in the G1 fractions from these animals with or without vanadate indicated the presence of vanadate-sensitive Tyr phosphatase in each G1 fractions. Mild tosylphenylalanyl chloromethyl ketone-trypsin treatment of the phosphorylated .alpha.-chain completely abolished PY with little detectable change in the mobility of the .alpha.-chains. These data indicate that a reversible Tyr phosphorylation occurs at N-terminal domain of the .alpha.-chain from these animals. To investigate whether the phosphorylation occurs in vivo, minced rat, rabbit and pig stomach tissues were incubated with pervanadate (PV), which permits the detection of Tyr phosphorylation through the irreversible inhibition. The results obtained indicate that a c-Src kinase present in the G1 membrane phosphorylates the N-terminal domain of the H/K-ATPase .alpha.-chain.

RE.CNT 12

RE

(1) Chew, C; Am J Physiol 1980, V238, PG312 CAPLUS
(2) Chew, C; Am J Physiol 1994, V267, PG818 CAPLUS
(3) Chew, C; Biochim Biophys Acta 1986, V888, P116 CAPLUS
(4) Chiba, T; Am J Physiol 1988, V255, PG99 CAPLUS
(5) Hersey, S; Biochim Biophys Acta 1983, V755, P293 CAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 5 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 3

AN 2000:157078 BIOSIS

DN PREV200000157078

TI Expression of extracellular matrix proteins in the fetal rat ***gastric*** mucosa.

AU Tommeras, Karin (1); Cabero, Jose Luis; ***Mardh, Sven***

CS (1) Department of Biomedicine and Surgery, Division of Cell Biology, Faculty of Health Sciences, Linkoping University, S-581 85, Linkoping Sweden

SO Anatomy and Embryology., (March, 2000) Vol. 201, No. 3, pp. 149-156. ISSN: 0340-2061.

DT Article

LA English

SL English

AB At gestational day 16 the epithelium of the rat stomach consists of a stratified layer of undifferentiated cells, and two days later glandular structures appear. The present study was carried out to identify extracellular matrix proteins that could be involved in the epithelial cell proliferation and differentiation processes that occur in the fetal rat stomach during this period. For comparative purposes the expression of the same components in the adult ***gastric*** mucosa was examined. Pregnant Sprague-Dawley rats received an intraperitoneal injection of 5-bromo-2'-deoxyuridine to label proliferating cells. One, 3.5, or 6 h post-injection the stomachs were excised and immediately frozen. The

specimens were sectioned and stained with hematoxylin and eosin or for 5-bromo-2'-deoxyuridine, cytokeratin no. 8, H,K-ATPase, and the extracellular matrix proteins fibronectin, laminin, and collagens type I and IV. A stratified layer of proliferating cells was observed in the epithelium of the fetal stomachs, while in adult stomachs proliferating cells were detected in the isthmus/neck region of the glands. Cytokeratin, an epithelial cell marker, was sparse at gestational day 16 but abundant both at gestational day 18 and in the isthmus/neck region of ***gastric*** glands of the adult stomach. The parietal cell marker H,K-ATPase could not be detected in the fetal stomachs during this period. Fibronectin was observed in the stroma of both fetal and adult stomachs. Collagen type I could only be detected in the stroma close to the oesophagus at gestational day 16. Two days later, collagen type I was abundant in the lamina propria, the submucosa and in the serosa of the fetal stomachs. In adult tissue collagen type I was detected in the surface epithelium, the submucosa and in the serosa of the stomach. Collagen type IV and laminin were expressed in the lamina propria, the basement membranes around blood vessels, muscle cells, and nerve bundles, as well as in the serosa of both 16- and 18-day-old fetal and adult rat stomachs. In conclusion, a high cell proliferation rate was observed in the epithelium at both gestational days 16 and 18. The increased expression of cytokeratin observed during this period indicates that the epithelial character of the embryonic cells becomes more distinct, while the remarkable change in the expression of collagen type I might reflect an important role of collagen type I in the development of the ***gastric*** epithelium.

L2 ANSWER 6 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 4
AN 2000:229681 BIOSIS
DN PREV200000229681
TI Helicobacter ***pylori*** -antigen-binding fragments expressed on the filamentous M13 phage prevent bacterial growth.
AU Cao, Jun; Sun, Yi-qian; Berglindh, Thomas; Mellgard, Bjorn; Li, Zhao-qi; Mardh, Bibbi; ***Mardh, Sven (1)***
CS (1) Department of Biomedicine and Surgery, Division of Cell Biology, Faculty of Health Sciences, Linkoping University, Linkoping Sweden
SO Biochimica et Biophysica Acta, (March 6, 2000) Vol. 1474, No. 1, pp. 107-113.
ISSN: 0006-3002.
DT Article
LA English
SL English
AB Colonization of the human stomach by Helicobacter ***pylori*** is associated with the development of ***gastritis*** , duodenal ulcer, mucosa-associated lymphoid tissue (MALT) lymphoma, and ***gastric*** cancer. H. ***pylori*** -antigen-binding single-chain variable fragments (ScFv) were derived from murine hybridomas producing monoclonal antibodies and expressed as a g3p-fusion protein on a filamentous M13 phage. The recombinant ScFv-phage reacted specifically with a 30-kDa monomeric protein of a H. ***pylori*** surface antigen preparation and by means of immunofluorescence microscopy the phage was shown to bind to both the spiral and coccoid forms of the bacterium. In vitro, the recombinant phage exhibited a bacteriocidal effect and inhibited specifically the growth of all the six strains of H. ***pylori*** tested. When H. ***pylori*** was pretreated with the phage 10 min

before oral inoculation of mice, the colonization of the mouse stomachs by the bacterium was significantly reduced ($P < 0.01$). The results suggest that genetic engineering may be used to generate therapy-effective phages.

L2 ANSWER 7 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 5
AN 2000:337801 BIOSIS
DN PREV200000337801
TI Effects of cholecystokinin on acid formation in glands and cells isolated from rabbit and rat ***gastric*** mucosa.
AU Bengtsson, Per (1); Azerkan, Leila; Lundqvist, Gudmar; Nilsson, Goran; ***Mardh, Sven***
CS (1) Department of Biomedicine and Surgery, Faculty of Health Sciences, Linkoping University, S-581 85, Linkoping Sweden
SO Comparative Biochemistry and Physiology Part A Molecular & Integrative Physiology, (May, 2000) Vol. 126A, No. 1, pp. 77-84. print.
ISSN: 1095-6433.
DT Article
LA English
SL English
AB Isolated ***gastric*** glands and isolated cells prepared from rabbit and rat were studied to analyse the influence of cholecystokinin octapeptide (CCK 8) on histamine stimulated parietal cell acid formation as assessed by (14C)aminopyrine sequestered in acid tissue compartments. In rabbit ***gastric*** glands, CCK 8 evoked 32+-6% ($P < 0.01$) inhibition of histamine stimulated acid formation, whereas in glands prepared from rat no inhibition was recorded. Instead, CCK 8 seemed to induce a variable increase of the histamine stimulation in rat ***gastric*** glands as the aminopyrine accumulation was increased by 110+-46% ($P < 0.1$). Further studies on cell preparations derived from rabbit ***gastric*** mucosa revealed dual properties of CCK 8, eliciting either inhibition or stimulation of the parietal cell depending on the presence of endocrine cells. The results show that paracrine communication may be effective in glandular preparations, but seems to vary depending on species.

L2 ANSWER 8 OF 86 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 1999:445434 SCISEARCH
GA The Genuine Article (R) Number: 187GJ
TI Expression of CCK-B/ ***gastrin*** receptors in undifferentiated epithelial cells of fetal rat stomachs.
AU Tommeras K (Reprint); Cabero J L; Forssmann W G; ***Mardh S***
CS FAC HLTH SCI, DEPT BIOMED & SURG, LINKOPING, SWEDEN; ASTRA HASSLE AB, DEPT BIOCHEM & CELL BIOL, MOLNDAL, SWEDEN; LOWER SAXONY INST PEPTIDE RES, HANNOVER, GERMANY
CYA SWEDEN; GERMANY
SO GASTROENTEROLOGY, (APR 1999) Vol. 116, No. 4, Part 2, pp. G2842-G2842.
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399.
ISSN: 0016-5085.
DT Conference; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 0

L2 ANSWER 9 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS

AN 1999:314082 BIOSIS
DN PREV199900314082
TI Expression of CCK-B/ ***gastrin*** receptors in undifferentiated epithelial cells of fetal rat stomachs.
AU Tommeras, K. (1); Cabero, J. L.; Forssmann, Wolf-Georg; ***Mardh, S.***
CS (1) Dept of Biomed and Surg, Faculty of Health Sci, Linkoping Sweden
SO Gastroenterology, (April, 1999) Vol. 116, No. 4 PART 2, pp. A652. Meeting Info.: Digestive Disease Week and the 100th Annual Meeting of the American Gastroenterological Association Orlando, Florida, USA May 16-19, 1999 American Gastroenterological Association . ISSN: 0016-5085.
DT Conference
LA English

L2 ANSWER 10 OF 86 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 1999:644918 SCISEARCH
GA The Genuine Article (R) Number: 226GJ
TI Direct evidence for in vivo reversible tyrosine phosphorylation of the N-terminal domain of the H/K-ATPase alpha-subunit in mammalian stomach cells
AU Kanagawa M; Kaya S; Umezu H; Watanabe S; Togawa K; Shimada A; Imagawa T; ***Mardh S*** ; Taniguchi K (Reprint)
CS HOKKAIDO UNIV, GRAD SCH SCI, SAPPORO, HOKKAIDO 060081, JAPAN (Reprint); HOKKAIDO UNIV, GRAD SCH SCI, SAPPORO, HOKKAIDO 060081, JAPAN; LINKOPING UNIV, FAC HLTH SCI, DEPT BIOMED & SURG, S-58185 LINKOPING, SWEDEN
CYA JAPAN; SWEDEN
SO JOURNAL OF BIOCHEMISTRY, (AUG 1999) Vol. 126, No. 2, pp. 266-270. Publisher: JAPANESE BIOCHEMICAL SOC, ISHIKAWA BLDG-3F, 25-16 HONGO-5-CHOME, BUNKYO-KU, TOKYO 113, JAPAN. ISSN: 0021-924X.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 29
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB In vivo reversible phosphorylation of Tyr-7 and Tyr-10 of the pig stomach H/K-ATPase alpha-chain was initially demonstrated in mammals, rat, rabbit, and pig, in the presence of vanadate+H2O2. In vitro phosphorylation has also been unequivocally demonstrated via the use of protease inhibitors during membrane H/K-ATPase preparation. An amphoretic detergent permitted each intrinsic kinase to phosphorylate each fusion protein containing the requisite Tyr residues, along with a reduction in alpha-chain phosphorylation. These and other data suggest that some important enzyme systems are present in the apical membrane and that they are in sufficient proximity to participate in the reversible phosphorylation of the amino terminal soluble domain of the alpha-chain with an unknown physiological function in the membrane embedded H/K-ATPase.

L2 ANSWER 11 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 6
AN 1998:258345 BIOSIS
DN PREV199800258345
TI Asymptomatic Helicobacter ***pylori*** ***gastritis*** is associated with increased sucrose permeability.
AU Borch, Kurt (1); Sjostedt, Camilla; Hannestad, Ulf; Soderholm, Johan D.;

Franzen, Lennart; ***Mardh, Sven***
CS (1) Dep. of Surgery, University Hospital, S-58185 Linkoping Sweden
SO Digestive Diseases and Sciences, (April, 1998) Vol. 43, No. 4, pp. 749-753.
ISSN: 0163-2116.
DT Article
LA English
AB Our aim was to investigate whether there are changes in permeability to sucrose in asymptomatic Helicobacter ***pylori*** ***gastritis*** . Nineteen asymptomatic subjects with Helicobacter ***pylori*** associated ***gastritis*** with no or mild mucosal atrophy and 19 age- and sex-matched normal controls were studied by peroral load of sucrose (100 g). The fraction of the given oral dose of sucrose excreted in urine was increased in subjects with Helicobacter ***pylori*** ***gastritis*** (median 0.08% versus 0.04% in controls). Sucrose excretion was not related to atrophy, intestinal metaplasia, or inflammation in the ***gastric*** mucosa. However, sucrose permeability was related to the degree of inflammatory (neutrophil) activity, since moderate activity was associated with higher sucrose excretion than mild activity (median 0.13% vs 0.07%). Asymptomatic Helicobacter ***pylori*** ***gastritis*** was associated with an increased sucrose permeability, which could be a sign of ***gastric*** mucosal leakage. This could have implications for the diseases and complications associated with Helicobacter ***pylori*** infection.

L2 ANSWER 12 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 7
AN 1998:405941 BIOSIS
DN PREV199800405941
TI Identification of Helicobacter in ***gastric*** biopsies by PCR based on 16S rDNA sequences: A matter of little significance for the prediction of H. ***pylori*** -associated ***gastritis*** .
AU Tiveljung, Annika; Borch, K.; Jonasson, J.; ***Mardh, S.*** ; Petersson, F.; Monstein, H.-J. (1)
CS (1) Div. Clinical Microbiol., Fac. Health Sci., Univ. Linkoping, Linkoping Sweden
SO Journal of Medical Microbiology, (Aug., 1998) Vol. 47, No. 8, pp. 695-704.
ISSN: 0022-2615.
DT Article
LA English
AB The aim of the present study was to correlate molecular evidence of the presence of Helicobacter ***pylori*** in ***gastric*** biopsy samples, based on analysis of 16S rDNA, vacuolating toxin (vacA), urease A (ureA) and cagA genes, with the clinical, histological and serological findings in patients with H. ***pylori*** -associated ***gastritis*** . Fresh biopsy samples were collected from the ***gastric*** antrum and corpus of 22 asymptomatic volunteers with or without H. ***pylori*** -associated ***gastritis*** . Total DNA was extracted from the biopsy material and subjected to 16S rDNA PCR amplification, Southern blotting and 16S rDNA sequence analysis of the PCR products. The vacA, ureA and cagA genes were characterised by PCR amplification and Southern blot analysis. Based on partial 16S rDNA sequence analysis, DNA belonging to the genus Helicobacter was detected in ***gastric*** biopsy samples from 20 of 22 subjects, including seven of nine histologically and serologically normal controls. Six of 20 partial 16S rDNA sequences revealed variations within variable regions V3 and V4 that deviated from

those of the H. ***pylori*** type strain ATCC 4350T and, therefore, possibly represented other species of Helicobacter. VacA genes identical with those of the type strain were found predominantly in the subjects with H. ***pylori*** ***gastritis*** , and all the patients except one were found to be cagA-positive. There was no evidence of false positive PCR reactions. In conclusion, the PCR-based molecular typing methods used here were apparently too sensitive when applied to the detection of H. ***pylori*** in human ***gastric*** tissues. The lack of quantitative analysis makes them inappropriate as clinical tools for the diagnosis of H. ***pylori*** -associated ***gastritis*** , despite the fact that they provide a qualitative and sensitive tool for the detection and characterisation of H. ***pylori*** in the gastrointestinal tract.

L2 ANSWER 13 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 8
AN 1998:118862 BIOSIS
DN PREV199800118862
TI Protein kinase-dependent phosphorylation of H,K ATPase-containing membranes from rat and pig stomachs.
AU Kaya, S. (1); ***Mardh, S.***
CS (1) Biol. Chem., Dep. Chem., Grad. Sch. Sci., Hokkaido Univ., Sapporo 060 Japan
SO Acta Physiologica Scandinavica, (Jan., 1998) Vol. 162, No. 1, pp. 57-62. ISSN: 0001-6772.
DT Article
LA English
AB Previously H, K ATPase preparations from pig stomach were shown to contain intrinsic protein kinase activities which phosphorylated specific tyrosine and serine residues in the N-terminal of the alpha-chain of H,K ATPase (Togawa et at. 1996). In the present investigation, pig H,K ATPase-containing membrane preparations were compared with rat preparations. In contrast to results obtained with the alpha-subunit of H,K ATPase from pig, phosphorylation was not observed in the rat enzyme. Addition of rat preparations to the pig preparations resulted in decreased phosphorylation in pig preparations. To follow the phosphorylation of membrane proteins in vivo, 32P-loaded ***gastric*** cells prepared from rat were stimulated with several secretagogues. Proteins with molecular weights of about 120 and 80 kDa were markedly phosphorylated upon stimulation, but the alpha-subunit of H,K ATPase was not. These results suggest that phosphorylation of tyrosine or serine residues of H,K ATPase found in pig H,K ATPase preparations may not be involved in the acid secretion pathway.

L2 ANSWER 14 OF 86 CAPLUS COPYRIGHT 2001 ACS
AN 1997:542514 CAPLUS
DN 127:186628
TI Bacteriophages presenting recombinant surface protein fusion products with bacteria antigen-binding antibody for use in bacterial infection treatment
IN ***Mardh, Sven***
PA Mardh, Sven, Swed.
SO PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 9729185 A1 19970814 WO 1997-SE172 19970205
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG
SE 9600434 A 19970807 SE 1996-434 19960206
SE 506771 C2 19980209
CA 2244792 AA 19970814 CA 1997-2244792 19970205
AU 9716817 A1 19970828 AU 1997-16817 19970205
AU 712767 B2 19991118
EP 889955 A1 19990113 EP 1997-902815 19970205
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
CN 1210558 A 19990310 CN 1997-192116 19970205
JP 2000505648 T2 20000516 JP 1997-528446 19970205
NO 9803456 A 19981006 NO 1998-3456 19980727
PRAI SE 1996-434 19960206
WO 1997-SE172 19970205

AB The present invention relates to bacteriophages for use in the treatment or prophylaxis of bacterial infections, esp. mucosal bacterial infections such as Helicobacter ***pylori*** infections. In particular, it relates to modified filamentous bacteriophages, e.g., M13 phages, for such use, which bacteriophages present at its surface a recombinant protein comprising: (i) a first component derived from a bacteriophage surface protein; and (ii) a second component comprising variable region sequences of an antibody to provide a bacterial antigen binding site, said second component rendering said bacteriophage capable of binding to and thereby inhibiting growth of bacterial cells involved in the etiol. of said infection.

L2 ANSWER 15 OF 86 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.DUPLICATE 9
AN 1998004376 EMBASE
TI Phosphorylation of Tyr7, Tyr10, and Ser27 of .alpha.-chain in H+,K+-ATPase by intrinsic and extrinsic kinases.
AU Togawa K.; Kaya S.; Mori M.; Shimada A.; Imagawa T.; Taniguchi K.; ***Mardh S.*** ; Corbin J.; Kikkawa U.
CS K. Taniguchi, Biological Chemistry, Graduate School of Science, Hokkaido University, Sapporo 060, Japan. KTAN@hucc.hokudai.ac.jp
SO Annals of the New York Academy of Sciences, (1997) 834/- (582-584).
Refs: 10
ISSN: 0077-8923 CODEN: ANYAA
CY United States
DT Journal; Conference Article
FS 029 Clinical Biochemistry
LA English

L2 ANSWER 16 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 10
AN 1998:95114 BIOSIS
DN PREV199800095114

TI Detection of spiral and coccoid forms of Helicobacter ***pylori***
using a murine monoclonal antibody.
AU Cao, Jun; Li, Zhao Q.; Borch, Kurt; Petersson, Fredrik; ***Mardh, Sven***
*** (1)***
CS (1) Div. Cellbiol., Dep. Biomed. Surg., Fac. Health Sci., Linkoping Univ.,
S-581 85 Linkoping Sweden
SO Clinica Chimica Acta, (Nov. 28, 1997) Vol. 267, No. 2, pp. 183-196.
ISSN: 0009-8981.
DT Article
LA English
AB Helicobacter ***pylori*** is the major cause of ***gastritis*** .
The aim of this investigation was to develop a specific antibody, which
recognizes both coccoid and spiral forms of Helicobacter ***pylori***
and to test this antibody on ***gastric*** biopsy sections known to
harbour coccoid bacteria. Murine monoclonal antibodies against
glycine-acid extracts of five strains of Helicobacter ***pylori***
were raised. Immunofluorescence and immunoelectron microscopy showed that
one antibody of the IgG1 subclass was specific for both the spiral and
coccoid forms. It reacted with a 28 kDa protein that was present in all
the five strains tested. Using this antibody in an indirect
immunofluorescence assay of formalin-fixed antral and corpus biopsy
specimens from Helicobacter ***pylori*** -associated ***gastritis***
patients showed that nine of the nine antral and five of six corpus
specimens harboured the coccoid form of Helicobacter ***pylori*** .
This technique thus provides a rapid and specific detection of both the
spiral and coccoid forms.

L2 ANSWER 17 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 11
AN 1997:129289 BIOSIS
DN PREV199799421102
TI Proliferation and differentiation of cells from explants of fetal rat
stomach.
AU Tommeras, K.; Chen, Y.; Rhedin, M.; Cabero, J. L.; ***Mardh, S. (1)***
CS (1) Dep. Cell Biol., Fac. Health Sci., Linkoping Univ., S-581 85 Linkoping
Sweden
SO Acta Physiologica Scandinavica, (1997) Vol. 159, No. 2, pp. 155-161.
ISSN: 0001-6772.
DT Article
LA English
AB The current understanding of the mechanisms controlling the proliferation
and differentiation of the stem cells of the ***gastric*** oxyntic
glands is limited. The aim of the present study was to develop a method
for investigating proliferation and differentiation of undifferentiated
cells from fetal rat stomach. Outgrowth of cells was initiated from
explants of 16-day-old fetal rat stomachs. At this stage of the fetal
development the ***gastric*** epithelial cells are undifferentiated.
The explants were cultured in DMEM/F-12 medium supplemented with fetal
calf serum only, or fetal calf serum combined with either hydrocortisone
or pentagastrin. Morphological characterization by means of light
microscopy, dye staining and immunostaining was used to identify the
growing cells. Both hydrocortisone and pentagastrin accelerated the
differentiation towards H,K-ATPase-positive cells, mucus-producing cells
and other epithelial cells. H,KATPase-positive cells, which were
identified by immunostaining with a monoclonal antibody reacting with the
alpha-subunit of the H,K-ATPase, grew on top of the confluent layer of

epithelioid and fibroblastoid cells. With this method in vitro investigations of the mechanisms of proliferation and differentiation of ***gastric*** mucosal cells are possible. Although by different mechanisms, both hydrocortisone and pentagastrin appear to play a regulatory role in these processes.

L2 ANSWER 18 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 12
AN 1997:277254 BIOSIS
DN PREV199799576457
TI Detection of spiral and coccoid forms of Helicobacter ***pylori*** using a murine monoclonal antibody.
AU Cao, J.; Li, Z.-Q.; Borch, K.; ***Mardh, S.***
CS Dep. Cell Biol., Fac. Health Sci., Linkoping Sweden
SO Gastroenterology, (1997) Vol. 112, No. 4 SUPPL., pp. A83.
Meeting Info.: Digestive Disease Week and the 97th Annual Meeting of the American Gastroenterological Association Washington, D.C., USA May 11-14, 1997
ISSN: 0016-5085.
DT Conference; Abstract
LA English

L2 ANSWER 19 OF 86 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 96:789662 SCISEARCH
GA The Genuine Article (R) Number: VP322
TI SER-27, TYR-10 AND TYR-7 IN THE ALPHA-CHAIN OF PIG STOMACH H+,K+-ATPASE AS CA2+-DEPENDENT PHOSPHORYLATABLE SITES BY INTRINSIC AND EXTRINSIC PROTEIN-KINASES
AU TOGAWA K; KAYA S; SHIMADA A; IMAGAWA T; ***MARDH S*** ; CORBIN J; KIKKAWA U; TANIGUCHI K (Reprint)
CS HOKKAIDO UNIV, GRAD SCH SCI, SAPPORO, HOKKAIDO 060, JAPAN (Reprint); HOKKAIDO UNIV, GRAD SCH SCI, SAPPORO, HOKKAIDO 060, JAPAN; LINKOPING UNIV, DEPT CELL PHYSIOL, S-58183 LINKOPING, SWEDEN; VANDERBILT UNIV, DEPT MOL PHYSIOL & BIOPHYS, NASHVILLE, TN, 37232; KOBE UNIV, BIOSIGNAL RES CTR, KOBE 657, JAPAN
CYA JAPAN; SWEDEN; USA
SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (23 OCT 1996) Vol. 227, No. 3, pp. 810-815.
ISSN: 0006-291X.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 16
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB When pig stomach membrane H+,K+-ATPase preparations were incubated with [gamma-P-32]ATP, Mg2+ and Ca2+, reversible phosphorylation of specific Tyr and Ser residues in the N-terminal alpha-chain of H+,K+-ATPase occurred without any detectable phosphorylation in other regions of the alpha-chain. Mild tosylphenyla lanyl chloromethyl ketone-trypsin treatment followed by reverse;phase column chromatography yielded three radioactive peptide peaks. The first peak contained both Tyr(10)(P-32) and Tyr(7)(P-32) and the second peak contained Tyr(10)(P-32). The third peak contained Ser(27)(P-32) which was also obtained after trypsin treatment of partially purified H-,K+-ATPase preparations phosphorylated with protein kinase-C + Ca2+ or protein kinase-A. This is the first demonstration of Ca2+-dependent phosphorylation of the alpha-chain of H+,K+-ATPase by

protein kinases. (C) 1996 Academic Press, Inc.

L2 ANSWER 20 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 13
AN 1996:313659 BIOSIS
DN PREV199699036015
TI Interactions between Ca-2+- and cAMP-dependent stimulatory pathways in parietal cells.
AU Li, Zhao-Qi; ***Mardh, Sven (1)***
CS (1) Dep. Cell Biol., Fac. Health Sci., Linkoping Univ., S-581 85 Linkoping Sweden
SO Biochimica et Biophysica Acta, (1996) Vol. 1311, No. 2, pp. 133-142.
ISSN: 0006-3002.
DT Article
LA English
AB Isolated rat parietal cells were used to investigate the role of intracellular Ca-2+ in the action of cAMP-dependent secretagogues and cross talk between cAMP- and Ca-2+-dependent stimulatory pathways. Aminopyrine accumulation (an index of acid produced and trapped by the parietal cells), cytosolic free Ca-2+, morphological transformation and cell viability were used to investigate parietal cell function 1,2-bis(2-aminophenoxy)ethane-N,N,N',N'-tetraacetic acid (BAPTA, 10 mu-M). Also, the morphological transformations induced by and stimulation. The increase of cytosolic free Ca-2+ promoted by ***gastrin*** , or carbachol, was abolished by the intracellular Ca-2+ chelator dibutytyladenosine 3':5'-cyclic monophosphate (DBcAMP), ***gastrin*** , and Sp-adenosine-cyclic-3',5'-monophosphothioate, (Sp-CAMPS) were completely abolished by BAPTA (10 mu-M). In aminopyrine accumulation the action of 1 mM DBcAMP was dose-dependently reduced by BAPTA. The Ca-2+ ionophore A23187 alone, in the range of 1 pM to 1 mu-M, had no effect but it dose-dependently potentiated the action of 1 mM DBcAMP in aminopyrine accumulation. The inhibitory actions of BAPTA on DBcAMP- and histamine-stimulated aminopyrine accumulation were dose-dependently reversed by A23187. Histamine-stimulated protein kinase activity and viability parameters as cellular lactate dehydrogenase (LDH) and trypan blue exclusion were not changed by BAPTA. These results indicated that in isolated parietal cells: (1) the action of cAMP-dependent secretagogues in aminopyrine accumulation and morphological transformation are dependent on cytosolic free Ca-2+; (2) Ca-2+-induced morphological transformation is essential for aminopyrine accumulation; (3) a threshold level of one second messenger is required for stimulation of aminopyrine accumulation by the other second messenger.

L2 ANSWER 21 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 14
AN 1995:154706 BIOSIS
DN PREV199598169006
TI ***Gastrin*** and carbachol require cAMP to elicit aminopyrine accumulation in isolated pig and rat parietal cells.
AU Li, Zhao-Qi; Cabero, Jose Luis; ***Mardh, Sven (1)***
CS (1) Dep. Cell Biol., Fac. Health Sci., Linkoping University, S-581 85 Linkoping Sweden
SO American Journal of Physiology, (1995) Vol. 268, No. 1 PART 1, pp. G82-G89.
ISSN: 0002-9513.
DT Article
LA English

AB The role of endogenous adenosine 3',5'-cyclic monophosphate (cAMP) in the mechanisms of action of ***gastrin*** and carbachol on aminopyrine accumulation in isolated pig and rat parietal cells was investigated. In pig cells, pentagastrin (100 nM) alone stimulated aminopyrine accumulation, an action significantly reduced by the protein kinase A inhibitor Rp-adenosine 3',5'-cyclic monophosphothioate (Rp-cAMP(S); 100 mu-M). In rat cells, ***gastrin*** -17 (100 nM) was incapable of stimulating aminopyrine accumulation, but it potentiated the action of histamine (100 mu-M). Carbachol (10 mu-M) stimulated aminopyrine accumulation and potentiated the action of histamine, and its action was potentiated in a dose-dependent manner by Sp-adenosine 3',5'-cyclic monophosphothioate (SpcAMP(S); a cAMP analogue) in both species. The effect of carbachol was dose dependently reduced by Rp-cAMP(S). The basal cAMP in pig parietal cells was 3.5-fold higher than that in rat parietal cells. Histamine (100 mu-M) and 3-isobutyl-1-methylxanthine (IBMX; 100 mu-M) only slightly elevated the cAMP content (1.2- to 2.9-fold the basal level) in both pig and rat parietal cells. Their combination, however, increased the cAMP level by 8- to 38-fold, but it did not increase aminopyrine accumulation above that elicited by histamine alone. ***Gastrin*** did not alter the cAMP levels in parietal cells of either of the two species. Both ***gastrin*** and carbachol increased cytosolic free Ca-2+ in enriched pig and rat parietal cells. These results indicated that in isolated pig and rat parietal cells 1) secretagogues that elevate intracellular free Ca-2+, such as ***gastrin*** and carbachol, require a certain cAMP level to be effective in stimulating aminopyrine accumulation; 2) a further increase over a certain level of cAMP does not result in an increased aminopyrine accumulation; and 3) parallel activation of Ca-2+- and cAMP-dependent pathways appears to be necessary to effectively elicit aminopyrine accumulation.

L2 ANSWER 22 OF 86 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 95:80651 SCISEARCH
GA The Genuine Article (R) Number: QB678
TI ***GASTRIN*** AND CARBACHOL REQUIRE CAMP TO ELICIT AMINOPYRINE ACCUMULATION IN ISOLATED PIG AND RAT PARIETAL-CELLS
AU LI Z Q; CABERO J L; ***MARDH S (Reprint)***
CS LINKOPING UNIV, FAC HLTH SCI, DEPT CELL BIOL, S-58185 LINKOPING, SWEDEN (Reprint); LINKOPING UNIV, FAC HLTH SCI, DEPT CELL BIOL, S-58185 LINKOPING, SWEDEN
CYA SWEDEN
SO AMERICAN JOURNAL OF PHYSIOLOGY-GASTROINTESTINAL AND LIVER PHYSIOLOGY, (JAN 1995) Vol. 31, No. 1, pp. G82-G89.
ISSN: 0193-1857.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 35
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The role of endogenous adenosine 3',5'-cyclic monophosphate (cAMP) in the mechanisms of action of ***gastrin*** and carbachol on aminopyrine accumulation in isolated pig and rat parietal cells was investigated. In pig cells, pentagastrin (100 nM) alone stimulated aminopyrine accumulation, an action significantly reduced by the protein kinase A inhibitor Rp-adenosine 3',5'-cyclic monophosphothioate (Rp-cAMP[S]; 100 mu M), In rat cells, ***gastrin*** -17 (100 nM) was incapable of

stimulating aminopyrine accumulation, but it potentiated the action of histamine (100 mu M). Carbachol (10 mu M) stimulated aminopyrine accumulation and potentiated the action of histamine, and its action was potentiated in a dose-dependent manner by Sp-adenosine 3',5'-cyclic monophosphothioate (Sp-cAMP[S]; a cAMP analogue) in both species. The effect of carbachol was dose dependently reduced by Rp-cAMP[S]. The basal cAMP in pig parietal cells was 3.5-fold higher than that in rat parietal cells. Histamine (100 mu M) and 3-isobutyl-1-methylxanthine (IBMX; 100 mu M) only slightly elevated the cAMP content (1.2- to 2.9-fold the basal level) in both pig and rat parietal cells. Their combination, however, increased the cAMP level by 8- to 38-fold, but it did not increase aminopyrine accumulation above that elicited by histamine alone. ***Gastrin*** did not alter the cAMP levels in parietal cells of either of the two species. Both ***gastrin*** and carbachol increased cytosolic free Ca2+ in enriched pig and rat parietal cells. These results indicated that in isolated pig and rat parietal cells 1) secretagogues that elevate intracellular free Ca2+, such as ***gastrin*** and carbachol, require a certain cAMP level to be effective in stimulating aminopyrine accumulation; 2) a further increase over a certain level of cAMP does not result in an increased aminopyrine accumulation; and 3) parallel activation of Ca2+- and cAMP-dependent pathways appears to be necessary to effectively elicit aminopyrine accumulation.

L2 ANSWER 23 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 15
AN 1995:61051 BIOSIS
DN PREV199598075351
TI Positive Correlation between H,K-Adenosine Triphosphatase Autoantibodies and Helicobacter ***pylori*** Antibodies in Patients with Pernicious Anemia.
AU Ma, J.-Y.; Borch, K.; Sjostrand, S. E.; Janzon, L.; ***Mardh, S. (1)***
CS (1) Dep. Cell Biol., Fac. Health Sci., S-581 85 Linkoping Sweden
SO Scandinavian Journal of Gastroenterology, (1994) Vol. 29, No. 11, pp. 961-965.
ISSN: 0036-5521.
DT Article
LA English
AB Background: Helicobacter ***pylori*** is a major cause of ***gastritis*** , and the parietal cell H,K-adenosine triphosphatase (ATPase) is a major autoantigen in autoimmune atrophic corpus ***gastritis*** , which may eventually lead to pernicious anemia and/or neuropathy. Whether the bacterium induces the autoimmune response is unknown. Methods: By means of enzyme-linked immunosorbent assay the occurrence of antibodies against porcine H,K-ATPase and H. ***pylori*** was determined in sera from 30 patients with pernicious anemia. Results: All sera scored positive against H,K-ATPase, and 25 (83%) scored positive against H. ***pylori*** . The titers of antibodies against both antigen preparations inversely correlated with the duration of disease. A possible common epitope in the antigen preparations was tested with a competition assay. There was no indication of a common epitope in either human or porcine H,K-ATPase and H. ***pylori*** . Conclusions: There was a positive correlation and a high incidence of antibodies against H,K-ATPase and H. ***pylori*** in sera from patients with pernicious anemia. These antibodies recognized different epitopes.

L2 ANSWER 24 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS

AN 1994:286849 BIOSIS
DN PREV199497299849
TI Mechanisms of action of secretagogues in isolated pig and rat parietal cells.
AU Li, Z.-Q.; Cabero, J. L.; ***Mardh, S.***
CS Dep. Cell Biol., Fac. Health, Science, Linkoping Sweden
SO Gastroenterology, (1994) Vol. 106, No. 4 SUPPL., pp. A822.
Meeting Info.: 95th Annual Meeting of the American Gastroenterological Association New Orleans, Louisiana, USA May 15-18, 1994
ISSN: 0016-5085.
DT Conference
LA English

L2 ANSWER 25 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 16
AN 1994:496414 BIOSIS
DN PREV199497509414
TI Human ***gastric*** H,K-adenosine triphosphatase beta-subunit is a major autoantigen in atrophic corpus ***gastritis*** .
AU Ma, J.-Y.; Borch, K.; ***Mardh, Sven (1)***
CS (1) Dep. Cell Biol., Fac. Health Sci., S-581 85 Linkoping Sweden
SO Scandinavian Journal of Gastroenterology, (1994) Vol. 29, No. 9, pp. 790-794.
ISSN: 0036-5521.
DT Article
LA English
AB Background: Sera from patients with atrophic corpus ***gastritis*** with pernicious anemia frequently contain parietal cell autoantibodies. We have previously demonstrated that the human H,K-adenosine triphosphatase (H,K-ATPase) alpha-subunit constitutes a major autoantigen. The present study investigates whether the human H,K-ATPase beta-subunit is an autoantigen, too. Methods: The gene of the human beta-subunit was expressed in insect cells by a baculovirus expression system. The reactivity of sera from 42 patients towards the recombinant glycoprotein was analyzed by means of an enzyme-linked immunosorbent assay. Results: Thirty-nine of the 42 sera (93%) scored positive. Autoantibody binding in 41 sera (98%) was eliminated when unglycosylated beta-subunit was used as antigen, and antibody binding in the last serum was decreased by 30%. Conclusions: The results indicate that the beta-subunit is indeed a major autoantigen and that carbohydrates are involved in binding of the autoantibodies.

L2 ANSWER 26 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 17
AN 1994:286477 BIOSIS
DN PREV199497299477
TI H,K-ATPase autoantibodies and Helicobacter ***pylori*** antibodies in patients with pernicious anemia.
AU ***Mardh, S.*** ; Ma, J.-Y.; Janzon, L.; Borch, K.
CS Dep. Cell Biol., Fac. Health Sci., Linkoping Sweden
SO Gastroenterology, (1994) Vol. 106, No. 4 SUPPL., pp. A729.
Meeting Info.: 95th Annual Meeting of the American Gastroenterological Association New Orleans, Louisiana, USA May 15-18, 1994
ISSN: 0016-5085.
DT Conference
LA English

L2 ANSWER 27 OF 86 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.DUPLICATE 18
AN 94057929 EMBASE
DN 1994057929
TI Localization of a pernicious anaemia autoantibody epitope on the .alpha.-subunit of human H,K-adenosine triphosphatase.
AU Song Y.-H.; Ma J.-Y.; ***Mardh S.*** ; Liu T.; Sjostrand S.E.; Rask L.; Borch K.; Huang G.-C.; Barnett P.; McGregor A.M.; Banga J.P.
CS Department Cell Biology, Faculty of Health Sciences,S-581 85 Linkoping, Sweden
SO Scandinavian Journal of Gastroenterology, (1994) 29/2 (122-127). ISSN: 0036-5521 CODEN: SJGRA4
CY Norway
DT Journal; Article
FS 025 Hematology
048 Gastroenterology
LA English
SL English
AB Four cDNA fragments encoding different portions of the .alpha.-subunit of human H,K-adenosine triphosphatase (ATPase) were amplified by means of the polymerase chain reaction technique, ligated into the plasmid pGEX-2T, and expressed as glutathione S-transferase fusion proteins in Escherichia coli. The fragments A (residues 163-313), Ba (residues 360-797), Bb (residues 526-797), and C (residues 822-1031) together encompass 77% of the .alpha.-subunit and cover most of its cytosolic part. Ther reactivities of autoantibodies in the sera from patients with pernicious anaemia with the recombinant fusion proteins were analysed by immunoblotting. One autoantigen epitope was found in the NH2-terminal part of the Ba fragment - that is, between residues 360 and 525. No epitope was detected in the other fragments. The Ba fragment was cleaved off from the glutathione S-transferase fusion protein by the action of thrombin and was then further purified. By means of enzyme-linked immunosorbent assay, 28 of 42 sera (67%) from patients with pernicious anaemia were positive against the purified Ba fragment. The present results provide a final proof that the human H,K-ATPase .alpha.-subunit is a major autoantigen in the parietal cell and that the major epitope is located between residues 360 to 525 on the cytosolic side of the secretory membrane.

L2 ANSWER 28 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 19
AN 1993:184628 BIOSIS
DN PREV199395095078
TI Direct ***gastrin*** action on isolated rat parietal cells induces morphological transformations.
AU Li, Zhao-Qi; Cabero, Jose Luis; Nilsson, B. Ove; ***Mardh, Sven (1)***
CS (1) Uppsala Biomedical Centre, Box 575, S-751 23 Uppsala Sweden
SO Biochimica et Biophysica Acta, (1993) Vol. 1175, No. 3, pp. 250-256. ISSN: 0006-3002.
DT Article
LA English
AB In isolated rat parietal cells, a potentiating effect by ***gastrin*** of the stimulatory action of histamine and dibutyryl-cAMP (DBcAMP) on aminopyrine accumulation, an index of the acid formed and trapped by the cells, was recently reported by us (1991, Am. J. Physiol. 261, G621-G627). In the present study, this mechanism of action of ***gastrin*** was further investigated. Enriched parietal cells (apprxeq 65% parietal cells) were incubated under different conditions and processed for

electron microscopy. Morphometric analysis of the micrographs revealed that pentagastrin (100 nM) was as efficient as histamine (100 mu-M) in inducing the formation of vacuolar/canalicular spaces in the parietal cells. In the presence of the histamine H-2-receptor antagonist ranitidine, histamine was ineffective but pentagastrin and ***gastrin*** -17 (G17) maintained their capacity to induce the morphological transformations. By stimulation with pentagastrin plus histamine, the vacuolar/canalicular volume was 2-fold higher than by stimulation separately with each one of the secretagogues. G-17 (100 nM) alone was ineffective but potentiated the maximal (14C)aminopyrine accumulation obtained with 100 mu-M histamine in mucosal cells (apprxeq 25-35% parietal cells). Ranitidine blocked both histamine- and histamine plus G-17-stimulated aminopyrine accumulation. G-17 potentiated also the stimulation by 1 mM dibutyryl-cyclic AMP but this was not inhibited by ranitidine. Pentagastrin (100 nM) increased the basal (14C)glucose oxidation in mucosal cells by 30%. This increase was not blocked by ranitidine which, however, abolished the histamine-stimulated glucose oxidation. Incubation of the cells with pentagastrin plus histamine resulted in a glucose oxidation which equaled the sum of the values obtained by each one of the agents. These results indicate that ***gastrin*** , acting directly on the parietal cells, potentiates the action of histamine on aminopyrine accumulation by increasing the vacuolar/canalicular spaces, a process that is reflected in the metabolic activity of the cells. Thus a major effect of ***gastrin*** at the parietal cell level appears to be the induction of a morphology which is characteristic of stimulated cells rather than a direct activation of ion-transport mechanisms.

L2 ANSWER 29 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 20
AN 1993:386071 BIOSIS
DN PREV199396061371
TI ***Gastrin*** action on aminopyrine accumulation in isolated pig parietal cells requires cAMP.
AU Cabero, Jose Luis; Li, Zhao-Qi; ***Mardh, Sven (1)***
CS (1) Dep. Cell Biol., Fac. Health Sci., S-581 85 Linkoping Sweden
SO Biochimica et Biophysica Acta, (1993) Vol. 1177, No. 3, pp. 245-252. ISSN: 0006-3002.
DT Article
LA English
AB The mechanism of action of ***gastrin*** on pig parietal cells was investigated. The aminopyrine accumulation technique was used to estimate acid production in ***gastric*** mucosal cells, containing 10-20% parietal cells, and in enriched parietal cells, containing 65-95% parietal cells. The ***gastrin*** analogue pentagastrin stimulated aminopyrine accumulation in a dose-dependent fashion irrespective of the proportion of non-parietal cells present. The apparent EC-50 for pentagastrin was 5 nM and the maximally effective concentration was 100 nM. The histamine H-2-receptor antagonist ranitidine did not affect the action of pentagastrin. The stimulatory effects of various doses of histamine on aminopyrine accumulation in highly enriched parietal cells were potentiated by the inclusion of 100 nM pentagastrin in the incubation medium. In another series of experiments using mucosal cells, the action of effective doses of pentagastrin were potentiated by the phosphodiesterase inhibitor isobutylmethyl xanthine (IBMX), which alone elicited an aminopyrine accumulation equal to 50% of that obtained by 100

mu-M histamine. When ranitidine (100 mu-M) was included, the action of IBMX was almost completely abolished. However, the dose-response curve for pentagastrin in the presence of ranitidine plus IBMX was similar to that obtained in the absence of IBMX. Dibutyryl-cAMP (DBcAMP, 1 mM) in the presence of ranitidine (100 mu-M) also potentiated the action of all effective doses of pentagastrin on mucosal cells. The protein kinase A inhibitor Rp-cAMPS, present at 500 mu-M in the incubation medium, significantly reduced the action of each effective concentration of pentagastrin on aminopyrine accumulation in enriched parietal cells. These results in pig parietal cells were interpreted as indicative of: (i) an action of ***gastrin*** exerted directly on the parietal cells; (ii) elevation of intracellular cAMP having a permissive role in the action of ***gastrin*** on aminopyrine accumulation.

L2 ANSWER 30 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1993:310169 BIOSIS
DN PREV199345016694
TI ***Gastric*** hydrogen, potassium-ATPase and mRNA-level in the rat after long term treatment with omeprazole, lansoprazole or pantoprazole.
AU Fryklund, J. (1); Torven, A.; Stalbom, B.-M.; Cabero, J. L.; ***Mardh,*** *** S.*** ; Lundberg, L.
CS (1) Astra Hassle AB, Molndal Sweden
SO Gastroenterology, (1993) Vol. 104, No. 4 SUPPL., pp. A82.
Meeting Info.: 94th Annual Meeting of the American Gastroenterological Association Boston, Massachusetts, USA May 15-21, 1993
ISSN: 0016-5085.
DT Conference
LA English

L2 ANSWER 31 OF 86 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 93:261313 SCISEARCH
GA The Genuine Article (R) Number: KX957
TI ***GASTRIC*** H,K-ATPASE AND MESSENGER RNA-LEVELS IN THE RAT AFTER LONG-TERM TREATMENT WITH OMEPRAZOLE, LANSOPRAZOLE OR PANTOPRAZOLE
AU FRYKLUND J (Reprint); TORVEN A; STALBOM B M; CABERO J L; ***MARDH S*** ; LUNDBERG L
CS ASTRA HASSLE AB, MOLNDAL, SWEDEN; UNIV UPPSALA, DEPT MED & PHYSIOL CHEM, S-75105 UPPSALA, SWEDEN
CYA SWEDEN
SO GASTROENTEROLOGY, (APR 1993) Vol. 104, No. 4, Supp. S, pp. A82.
ISSN: 0016-5085.
DT Conference; Journal
FS LIFE; CLIN
LA ENGLISH
REC No References

L2 ANSWER 32 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 21
AN 1992:255992 BIOSIS
DN BA93:132317
TI EFFECTS OF ***GASTRIN*** ON CYTOSOLIC FREE CALCIUM IN INDIVIDUAL ACID-SECRETING RAT PARIETAL CELLS.
AU CABERO J L; GRAPENGIESSER E; GYLFE E; LI Z-Q; ***MARDH S***
CS DEP. MED. PHYSIOL. CHEM., BOX 575, UPPSALA UNIV., UPPSALA S-751 23, SWED.
SO BIOCHEM BIOPHYS RES COMMUN, (1992) 183 (3), 1097-1102.
CODEN: BBRCA9. ISSN: 0006-291X.

FS BA; OLD
LA English
AB The effects of ***gastrin*** on cytosolic free Ca2+ ([Ca2+]i) in single, isolated rat ***gastric*** parietal cells were investigated using the fluorescent probe Fura-2 and digital image analysis. [Ca2+]i was increased by ***gastrin*** (100 nM) in .apprxeq. 30% of the parietal cells, which were identified by using either the fluorescent probe acridine orange or a parietal cell-specific monoclonal antibody. In the dominant pattern observed, [Ca2+]i was elevated 50-150% and returned within 1-2 min to a value 30-60% over the basal, which was sustained until withdrawal of the stimulant or addition of the ***gastrin*** inhibitor L-365,260 (1 .mu.M). The second, but not the first phase, was abolished in the absence of extracellular Ca2+. The results indicate the existence of functional ***gastrin*** receptors in a subpopulation of rat parietal cells.

L2 ANSWER 33 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 22
AN 1992:235380 BIOSIS
DN BA93:123405
TI PRODUCTION OF MONOCLONAL ANTIBODIES AGAINST ***GASTRIC*** PARIETAL CELL ANTIGENS.
AU CABERO J L; SASAKI T; SONG Y-H; HOLMDAHL R; ***MARDH S***
CS DEP. MED. AND PHYSIOL. CHEM., BIOMED. CENTRE, UPPSALA UNIV., P.O. BOX 575, S-751 23 UPPSALA, SWEDEN.
SO ACTA PHYSIOL SCAND, (1992) 144 (3), 369-378.
CODEN: APSCAX. ISSN: 0001-6772.
FS BA; OLD
LA English
AB Two mice DBA/1 were each immunized with a single injection of one million enriched parietal cells in the hind foot pads. Monoclonal antibodies to be used as research tools in studies on regulatory mechanisms in ***gastric*** parietal cells were obtained after fusion of mouse myeloma cells (SP2) with cells from the popliteal lymph nodes of the mice. Twelve hybridomas produced antibodies reactive with structures only present in parietal cells as assessed by immunohistochemistry of oxyntic mucosa sections. Three hybridomas were subcloned and the antibodies produced by them, designated as PC4, PC8, and PC117, were characterized. In an enzyme-linked immunosorbent assay, all antibodies reacted with H,K-ATPase-containing vesicles. The antibody PC8 recognized a 94 kDa protein after immunoblotting of H,K-ATPase-containing vesicles and all antibodies precipitated a 94 kDa protein from [125I]H,K-ATPase-containing vesicles. The antibodies PC4 and PC117 recognized extracellular structures with a polarized distribution in viable, purified parietal cells. The results suggest that the structure recognized by all three antibodies is the .alpha.-subunit of the H,K-ATPase. The antibodies produced by another hybridoma, PC43, recognized a structure present in parietal and surface epithelial cells of the oxyntic mucosa. In an enzyme-linked immunosorbent assay, they reacted with a high-activity carbonic anhydrase which had been affinity-purified from pig oxyntic mucosa and they recognized a 30 kDa protein after immunoblotting. Thus, monoclonal antibodies against both intracellular and extracellular parietal cell structures were obtained after immunization with a small number of parietal cells.

L2 ANSWER 34 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 23
AN 1993:43717 BIOSIS

DN PREV199344020567
TI A continuous flow techniques for analysis of the stoichiometry of the ***gastric*** proton, potassium-ATPase.
AU ***Mardh, Sven (1)*** ; Norberg, L.
CS (1) Dep. Med. and Physiological Chem., Biomed. Centre, Uppsala Univ., Box 575, S-751 23 Uppsala
SO Acta Physiologica Scandinavica Supplementum, (1992) Vol. 0, No. 607, pp. 259-263.
Meeting Info.: Satellite Symposium of the 15th International Congress of Biochemistry on Ion Pumps, Structure and Mechanism, Gothenburg, Sweden, August 12-14, 1991. ACTA PHYSIOL SCAND SUPPL
ISSN: 0302-2994.
DT Article
LA English

L2 ANSWER 35 OF 86 MEDLINE
AN 93080083 MEDLINE
DN 93080083
TI A continuous flow technique for analysis of the stoichiometry of the ***gastric*** H,K-ATPase.
AU ***Mardh S*** ; Norberg L
CS Department of Medical and Physiological Chemistry, Uppsala University, Sweden..
SO ACTA PHYSIOLOGICA SCANDINAVICA. SUPPLEMENTUM, (1992) 607 259-63.
Journal code: 1UF. ISSN: 0302-2994.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199303
AB A continuous flow method was developed for determining the stoichiometry of the ***gastric*** proton pump H,K-ATPase in its hydrolysis of ATP, translocation of H+ and the K+ congener 86Rb+. H,K-ATPase-containing vesicles which had been isolated from pig ***gastric*** mucosa were incubated at 37 degrees C for 2 h in 150 mM 86RbCl, 0.5 mM EGTA and 3 mM Mes-buffer adjusted to pH 6.1 with Tris, and then applied to a 0.45 micron pore size filter. The immobilized vesicles were superfused with 0.15 mM Mes/Tris buffer, pH 6.1, containing 150 mM choline-Cl and 0.2 mM MgCl2. After the medium was changed to one containing 0.1 mM ATP, the amounts and rates of H+ uptake, 86Rb+ efflux, and ATP hydrolysis were measured in fractions collected after the filter. The initial ratio of transported Rb+ to hydrolysed ATP gave values of 0.96 +/- 0.26 (mean +/- SD, n = 28). The initial ratio of ATP-dependent Rb+ efflux to H+ uptake gave values of 0.92 +/- 0.28 (mean +/- SD, n = 28). The MgATPase activity was measured in vesicles which had been incubated with choline-Cl instead of RbCl. In the initial fractions used for calculation of the stoichiometry, the MgATPase activity was 15.8% +/- 8.7 (mean +/- S.D.) of the maximal ATPase activity obtained with Rb(+)-loaded vesicles. The MgATPase may be an intrinsic activity of the H,K-ATPase. However, whether corrections were made for the MgATPase or not, it had only marginal effects on the calculations of the stoichiometry of the pump.(ABSTRACT TRUNCATED AT 250 WORDS)

L2 ANSWER 36 OF 86 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 92:603727 SCISEARCH
GA The Genuine Article (R) Number: JT268

TI A CONTINUOUS-FLOW TECHNIQUE FOR ANALYSIS OF THE STOICHIOMETRY OF THE ***GASTRIC*** H,K-ATPASE
AU ***MARDH S (Reprint)*** ; NORBERG L
CS UNIV UPPSALA, CTR BIOMED, DEPT MED & PHYSIOL CHEM, BOX 575, S-75123 UPPSALA, SWEDEN (Reprint)
CYA SWEDEN
SO ACTA PHYSIOLOGICA SCANDINAVICA, (1992) Vol. 146, Supp. 607, pp. 259-263. ISSN: 0001-6772.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 12
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB A continuous flow method was developed for determining the stoichiometry of the ***gastric*** proton pump H,K-ATPase in its hydrolysis of ATP, translocation of H+ and the K+ congener Rb-86+. H,K-ATPase-containing vesicles which had been isolated from pig ***gastric*** mucosa were incubated at 37-degrees-C for 2 h in 150 mM (RbCl)-Rb-86, 0.5 mM EGTA and 3 mM Mes-buffer adjusted to pH 6.1 with Tris, and then applied to a 0.45 mum pore size filter. The immobilized vesicles were superfused with 0.15 mM Mes/Tris buffer, pH 6.1, containing 150 mM choline-Cl and 0.2 mM MgCl2. After the medium was changed to one containing 0.1 mM ATP, the amounts and rates of H+ uptake, Rb-86+ efflux, and ATP hydrolysis were measured in fractions collected after the filter. The initial ratio of transported Rb+ to hydrolysed ATP gave values of 0.96 +/- 0.26 (mean +/- SD, n = 28). The initial ratio of ATP-dependent Rb+ efflux to H+ uptake gave values of 0.92 +/- 0.28 (mean +/- SD, n = 28). The MgATPase activity was measured in vesicles which had been incubated with choline-Cl instead of RbCl. In the initial fractions used for calculation of the stoichiometry, the MgATPase activity was 15.8% +/- 8.7 (mean +/- S.D.) of the maximal ATPase activity obtained with Rb+-loaded vesicles. The MgATPase may be an intrinsic activity of the H,K-ATPase. However, whether corrections were made for the MgATPase or not, it had only marginal effects on the calculations of the stoichiometry of the pump. Thus, simultaneous measurements of Rb-86+ efflux, H+ uptake and ATP hydrolysis in immobilized ***gastric*** vesicles gave a stoichiometry of the pump close to a 1:1:1 ratio.

L2 ANSWER 37 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1993:107592 BIOSIS
DN PREV199344049992
TI Effects of ***gastrin*** on isolated rat parietal cells.
AU Li, Zhao-Qi; Cabero, Jose Luis; ***Mardh, Sven***
CS Dep. Med. and Physiological Chemistry, Uppsala Univ., Uppsala Sweden
SO Acta Physiologica Scandinavica Supplementum, (1992) Vol. 0, No. 608, pp. 160.
Meeting Info.: XX Nordic Congress of Physiology and Pharmacology Copenhagen, Denmark August 16-19, 1992
ISSN: 0302-2994.
DT Conference
LA English

L2 ANSWER 38 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 24
AN 1992:233887 BIOSIS
DN BA93:121912

TI TWO-DIMENSIONAL CRYSTALS OF MEMBRANE-BOUND ***GASTRIC*** PROTON POTASSIUM ATPASE.
AU HEBERT H; XIAN Y; HACKSELL I; ***MARDH S***
CS CENT. STRUCTURAL BIOCHEMISTRY, KAROLINSKA INST., NOVUM, S-141 57 HUDDINGE, SWED.
SO FEBS (FED EUR BIOCHEM SOC) LETT, (1992) 299 (2), 159-162.
CODEN: FEBLAL. ISSN: 0014-5793.
FS BA; OLD
LA English
AB Two-dimensional crystallization of membrane-bound H,K-ATPase (EC 3.6.1.36) in vesicle preparations from parietal cells of hog ***gastric*** mucosa was induced by an imidazole buffer containing Mg2+ and VO3- ions. A continuous reorganization of the protein molecules started within a few hours by the formation of linear arrays. At later stages confluent two-dimensional crystals were formed. Electron microscopy and image processing showed that these were of a single tetragonal type. The asymmetric unit consisted of one pear-shaped protein domain corresponding to a H,K-ATPase protomer. Through stain-deficient contact regions four adjacent protein units were connected forming a tetrameric structure.

L2 ANSWER 39 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 25
AN 1992:5300 BIOSIS
DN BA93:5300
TI OCCURRENCE OF AUTOANTIBODIES AGAINST INTRINSIC FACTOR PROTON POTASSIUM ATPASE AND PEPSINOGEN IN ATROPHIC ***GASTRITIS*** AND RHEUMATOID ARTHRITIS.
AU ***MARDH S*** ; MA J Y; SONG Y H; ALY A; HENRIKSSON K
CS DEP. MED. PHYSIOL. CHEM., BIOMED. CENTER, UPPSALA UNIV., BOX 575, S-751 23 UPPSALA, SWED.
SO SCAND J GASTROENTEROL, (1991) 26 (10), 1089-1096.
CODEN: SJGRA4. ISSN: 0036-5521.
FS BA; OLD
LA English
AB The occurrence of autoantibodies against intrinsic factor, H,K-ATPase, and pepsinogen was analysed by means of enzyme-linked immunosorbent assay in three groups of sera. Group 1 comprised sera from 14 rheumatoid arthritis patients with normal acid secretion; group 2, sera from 18 rheumatoid arthritis patients with reduced acid secretion; and group 3, sera from 11 patients with pernicious anaemia or achylia. Groups 1 and 2 were rheumatoid factor-positive, and group 3 was negative. Intrinsic factor autoantibodies were low in groups 1 and 2. In group 3, 9 of the 11 sera (82%) scored positive. The highest titres of H,K-ATPase and pepsinogen autoantibodies were found in groups 2 and 3. Only one serum in group 1 scored positive against H,K-ATPase, and two against pepsinogen, whereas corresponding values were 11 (61%) and 7 (39%) in group 2, and 10 (91%) and 6 (55%) in group 3. Autoantibodies against H,K-ATPase from a pool of patient sera recognized both the .alpha.- and .beta.-subunits of the enzyme. The present results support the hypothesis of an autoimmune disease overlap between non-organ-specific rheumatoid arthritis and organ-specific pernicious anaemia.

L2 ANSWER 40 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 26
AN 1992:25653 BIOSIS
DN BA93:14928
TI ***GASTRIN*** POTENTIATES HISTAMINE-STIMULATED AMINOPYRINE

ACCUMULATION IN ISOLATED RAT PARIETAL CELLS.
AU CABERO J L; LI Z-Q; ***MARDH S***
CS BIOMEDICAL CENTRE, UPPSALA UNIVERSITY, BOX 575, S-751 23 UPPSALA, SWED.
SO AM J PHYSIOL, (1991) 261 (4 PART 1), G621-G627.
CODEN: AJPHAP. ISSN: 0002-9513.
FS BA; OLD
LA English
AB Rat ***gastric*** mucosal cells, containing 25-35% parietal cells, were obtained by a modified isolation procedure involving protease, ethylene glycol-bis(.beta.-aminoethyl ether)-N,N,N',N'-tetraacetic acid, and mechanical treatments. Parietal cell responsiveness to secretagogues was assessed by the accumulation of the weak base [14C]aminopyrine in intracellular acidic compartments. Histamine, without phosphodiesterase inhibitors, dose dependently stimulated aminopyrine accumulation with an effective concentration producing 50% of maximal response of 13 .mu.M and a maximal effective dose of 100 .mu.M. Pentagastrin and rat ***gastrin*** -17 alone were ineffective but potentiated dose dependently the action of 100 .mu.M histamine. The mean potentiating effect varied from 32 to 70% for 100 nM pentagastrin and from 36 to 95% for 100 nM rat ***gastrin*** -17. Pentagastrin (100 nM) also potentiated the effect of 1 mM dibutyryrladenosine 3',5'-cyclic monophosphate (cAMP) by 44%, but it did not increase further the stimulation by carbachol. The potentiating effect of pentagastrin on histamine- and dibutyryl cAMP-stimulated aminopyrine accumulation was also observed after enrichment of parietal cells to 65-85%. The endogenous histamine was insufficient to stimulate acid production. Therefore ***gastrin*** appears to have a direct action also in rat parietal cells.

L2 ANSWER 41 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 27
AN 1991:362541 BIOSIS
DN BA92:50766
TI PEPTIC ULCER DISEASE ABSENCE OF ANTIBODIES STIMULATING THE HISTAMINE SENSITIVE ADENYLATE CYCLASE OF ***GASTRIC*** MUCOSAL CELLS.
AU BURMAN P; ***MARDH S*** ; LOOF L; NAESDAL J; KARLSSON F A
CS DEP. INTERNAL MEDICINE, UNIVERSITY HOSPITAL, S-751 85 UPPSALA, SWED.
SO GUT, (1991) 32 (6), 620-623.
CODEN: GUTTAK. ISSN: 0017-5749.
FS BA; OLD
LA English
AB The possible presence of parietal cell stimulating antibodies was examined in sera from 57 patients with relapsing ulcer disease. The sera were obtained at the time of symptomatic relapse and all patients had ulcers confirmed by endoscopy. A sensitive assay based on adenosine 3':5' cyclic monophosphte (cAMP) production in isolated procine ***gastric*** mucosal cells was used as a measure. cAMP production increased up to four hours of incubation and was histamine responsive; an approximately 20-fold increase was found with histmaine 10-4 mol/l. Sera from both patients and healthy control subjects showed some inhibitory effect on basal cAMP production compared with incubation in medium only, whereas immunoglobulin preparations had a weaker non-specific effect. No stimulation was found when the patients' sera and immunoglobulins (up to a concnetration of 6 mg/ml) were examined. These results that ***gastric*** acid hypersecretion in duodenal ulcer disease is not an effect of histamine receptor stimulating antibodies. The data thus argue against a recent hypothesis that severe chronic ulcer disease in some patients has an

autoimmune origin.

L2 ANSWER 42 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 28
AN 1992:468999 BIOSIS
DN BR43:90349
TI EFFECTS OF ***GASTRIN*** ON ISOLATED PARIETAL CELLS.
AU ***MARDH S*** ; CABERO J L; LI Z-Q
CS DEP. MED. PHYSIOLOGY CHEM., UPPSALA UNIV. BIOMEDICAL CENTRE, BOX 575, S-751 23 UPPSALA, SWED.
SO HAKANSON, R. AND F. SUNDLER (ED.). FERNSTROM FOUNDATION SERIES, VOL. 15. THE STOMACH AS AN ENDOCRINE ORGAN; 18TH ERIC K. FERNSTROM SYMPOSIUM, LUND, SWEDEN, MAY 21-23, 1990. XIX+548P. ELSEVIER SCIENCE PUBLISHERS B.V.: AMSTERDAM, NETHERLANDS; (DIST. IN THE USA AND CANADA BY ELSEVIER SCIENCE PUBLISHING CO., INC.: NEW YORK, NEW YORK, USA). ILLUS. (1991) 0 (0), 253-266.
CODEN: FFOSDF. ISSN: 0167-7004. ISBN: 0-444-81377-2.
DT Conference
FS BR; OLD
LA English

L2 ANSWER 43 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 29
AN 1992:4790 BIOSIS
DN BA93:4790
TI COMPLEMENTARY DNA CLONING OF THE BETA-SUBUNIT OF THE HUMAN ***GASTRIC*** PROTON POTASSIUM ATPASE.
AU MA J-Y; SONG Y-H; SJOSTRAND S E; RASK L; ***MARDH S***
CS DEP. MED. PHYSIOLOGICAL CHEM., BIOMED. CENTRE, BOX 575, S-751 23 UPPSALA, SWED.
SO BIOCHEM BIOPHYS RES COMMUN, (1991) 180 (1), 39-45.
CODEN: BBRCA9. ISSN: 0006-291X.
FS BA; OLD
LA English
AB A full-length cDNA clone encoding the human ***gastric*** H,K-ATPase (EC 3. 6. 1. 36) .beta.-subunit was isolated from a human ***gastric*** mucosal .lambda.gt10 library using oligonucleotide probes which were based on the cDNA sequence from rat and rabbit H,K-ATPase .beta.-subunits. The insert was 1407 bp in length and encoded a polypeptide of 291 amino acids with a MW = 33,367 Da. It exhibited 84.2%, 85.6% and 81.3% identity to the H,K-ATPase .beta.-subunits of rabbit, pig and rat, respectively.

L2 ANSWER 44 OF 86 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 91:511129 SCISEARCH
GA The Genuine Article (R) Number: GD854
TI EFFECTS OF ***GASTRIN*** ON ISOLATED RAT PARIETAL-CELLS
AU CABERO J (Reprint); LI Z Q; BANDYOPADHYAY S; ***MARDH S***
CS UNIV UPPSALA, CTR BIOMED, DEPT MED & PHYSIOL CHEM, S-75123 UPPSALA, SWEDEN
CYA SWEDEN
SO ACTA PHYSIOLOGICA SCANDINAVICA, (1991) Vol. 143, No. 1, pp. A15.
DT Conference; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 3

L2 ANSWER 45 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 30
AN 1991:112070 BIOSIS

DN BA91:59460
TI A CONTINUOUS-FLOW TECHNIQUE FOR ANALYSIS OF STOICHIOMETRY AND TRANSPORT KINETICS OF ***GASTRIC*** HYDROGEN POTASSIUM ATPASE.
AU NORBERG L; ***MARDH S***
CS DEP. MED. PHYSIOLOGICAL CHEM., BIOMEDICAL CENTRE, UPPSALA UNIV., BOX 575, S-751 23 UPPSALA, SWED.
SO ACTA PHYSIOL SCAND, (1990) 140 (4), 567-574.
CODEN: APSCAX. ISSN: 0001-6772.
FS BA; OLD
LA English
AB A continuous-flow method was developed for determining the stoichiometry of the ***gastric*** proton pump H,K-ATPase (EC 3.6.1.36) in its hydrolysis of ATP and translocation of H+ and the K+ congener 86Rb+. H,K-ATPase-containing vesicles which had been isolated from pig ***gastric*** mucosa were incubated at 37.degree. C for 2 h in 150 mM 86RbCl, 0.5 mM ethylenebis(oxyethylenenitrilo)tetra-acetic acid and 3 mM 2-(N-morpholino)ethane sulphonic acid (Mes) adjusted to pH 6.1 with Tris, and then applied onto a 0.45 .mu.m pore size cellulose acetate filter. The immobilized vesicles were superfused with 0.15 mM Mes/Tris buffer, pH 6.1, containing 150 mM choline chloride and 0.2 mM MgCl/2. After changing to a medium containing 0.1 mmM ATP, the amounts and rates of H+ uptake, 86Rb+ efflux and ATP hydrolysis were measured. The initial ratio of Rb+ transported to ATP hydrolysed gave values of 0.96 .+-. 0.26 (mean .+-. SD, n = 28). The initial ratio of ATP-dependent Rb+ efflux to H+ uptake gave values of 0.92 .+-. 0.28 (mean .+-. SD, n = 28). The Mg-ATPase activity was measured in vesicles which had been incubated with choline chloride instead of RbCl. This activity was 15.8 .+-. 8.7% (mean .+-. SD) of the total ATPase activity in the initial fractions used for calculation of the stoichiometry. It is argued that this Mg-ATPase may be an intrinsic activity of the H,K-ATPase and that the relation between these activities is dependent on the amount of K+ (or Rb+) present in the assay. However, whether corrections were made for this Mg-ATPase or not, it had only marginal effects on the calculations of the stoichiometry of the pump. Thus simultaneous measurements of 86Rb+ efflux, H+ uptake and ATP hydrolysis in immobilized ***gastric*** vesicles gave a stoichiometry of the pump close to a 1:1:1 ratio. These results indicate that the pump is non-electrogenic.

L2 ANSWER 46 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 31
AN 1991:47194 BIOSIS
DN BA91:25475
TI CALCIUM AND CALMODULIN STIMULATE PHOSPHOLIPASE A-2 AND FUSION OF HYDROGEN POTASSIUM ATPASE-CONTAINING MEMBRANE VESICLES ISOLATED FROM PIG ***GASTRIC*** MUCOSA.
AU OLAISSON H; ***MARDH S*** ; ARVIDSON G
CS DEP. MED. PHYSIOL. CHEM., BIOMED. CENT., UNIV. UPPSALA, BOX 575, S-751 23 UPPSALA, SWEDEN.
SO ACTA PHYSIOL SCAND, (1990) 140 (3), 393-400.
CODEN: APSCAX. ISSN: 0001-6772.
FS BA; OLD
LA English
AB Fusion of pig ***gastric*** H,K-ATPase- and phospholipase A2-containing vesicles in vitro was studied by electron microscopy and by monitoring the change in fluorescence of octadecyl rhodamine B-labelled vesicles. Ca2+ stimulated fusion of vesicles, and the fusion rate showed a

positive correlation with the activity of the phospholipase A2. Both the Ca2+-stimulated fusion rate and the Ca2+-dependent phospholipase A2 activity were futher enhanced by the presence of calmodulin. The present results supported our previous findings (Olaisson et al. 1990) and further indicate that the phospholipase A2 associated with the H,K-ATPase-containing membranes might play a central role in membrane fusion processes in the stimulated parietal cell.

L2 ANSWER 47 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 32
AN 1991:52202 BIOSIS
DN BA91:30483
TI OCCURRENCE OF PHOSPHOLIPASE A-2 AND LYSOPHOSPHOLIPASE IN A ***GASTRIC*** HYDROGEN POTASSIUM ATPASE-CONTAINING MEMBRANE FRACTION AND THE FORMATION OF LYSOPHOSPHATIDYLCHOLINE IN STIMULATED PIG PARIETAL CELLS.
AU OLAISSON H; ARVIDSON G; MA J-Y; ***MARDH S***
CS DEP. MED. PHYSIOL. CHEM., BIOMED. CENT., UPPSALA UNIV., BOX 575, S-751 23 UPPSALA, SWEDEN.
SO ACTA PHYSIOL SCAND, (1990) 140 (3), 383-392.
CODEN: APSCAX. ISSN: 0001-6772.
FS BA; OLD
LA English
AB A membrane fraction containing H,K-ATPase (EC 3.6.1.36) was prepared from pig ***gastric*** mucosa and found to contain phospholipase A2 (EC 3.1.1.4) and lysophospholipase (EC 3.1.1.5) activities. Washing the membranes decreased their protein content by 25%. Recovery profiles of H,K-ATPase, phospholipase A2 and lysophospholipase were similar for membranes washed either with water or with 0.15 or 1.5M KCl. Nearly identical distribution profiles were obtained for the three enzyme activities after centrifugation of washed vesicle membranes on a linear sucrose gradient. The phospholipase A2 activity was stimulated by calcium and increased further in the presence of calmodulin. The amount of cellular radioactively labelled lysophosphatidylcholine was doubled upon cholinergic stimulation of isolated parietal cells prelabelled with [3H]glycerol or 32P. The liberated lyso[32P]phosphatidylcholine had its acyl chain in the sn-1 position, which implies an activation of a phospholipase A2. These findings indicate that secretagogues which increase the cytosolic Ca2+ concentration, i.e. acetylcholine, histamine and ***gastrin*** , may activate a phospholipase A2 in the parietal cell.

L2 ANSWER 48 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 33
AN 1990:216332 BIOSIS
DN BA89:113622
TI BINDING OF CHOLECYSTOKININ AND SOMATOSTATIN TO ISOLATED PORCINE ***GASTRIC*** MUCOSAL CELLS AND EFFECTS ON AMINOPYRINE UPTAKE.
AU SJODIN L; ENGLUND L J; ***MARDH S***
CS SOCIALSTYRELSENS LAKEMEDELSAVDELNING, PO BOX 607, S-751 25 UPPSALA, SWED.
SO ACTA PHYSIOL SCAND, (1990) 138 (3), 369-376.
CODEN: APSCAX. ISSN: 0001-6772.
FS BA; OLD
LA English
AB Mucosal cells were prepared by enzymatic digestion of procine ***gastric*** mucosa with pronase and collagenase. The resulting cell suspension contained 10-15% parietal cells, which responded to histamine stimulation by an up to 20-fold increase in [14C]aminopyrine accumulation

over control levels. Cholecystokinin-8 (CCK-8) evoked a more moderate stimulation of [14C]aminopyrine accumulation, whereas somatostatin inhibited histamine-stimulated accumulation. Parietal cells were enriched by elutriation and isopycnic centrifugation on density gradients of Percoll. A fraction with 60% parietal cells bound approximately three times more iodinated CCK-8 than a fraction containing 70% non-parietal cells. Binding of [125I]BH-CCK-8 to preparations containing 30-60% parietal cells was specifically inhibited to about 50% by 10-9 M unlabelled CCK-8 but not by bombesin. Cell fractions containing about 30% parietal cells also bound [125I]somatostatin. Unlabelled somatostatin at 10-9 M inhibited tracer binding by about 50%, while CCK-8 did not affect somatostatin binding to such a preparation. The results suggest the existence of specific receptors for CCK and somatostatin on porcine parietal cells exerting a regulatory influence on acid secretion.

L2 ANSWER 49 OF 86 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 90233623 EMBASE
DN 1990233623
TI The occurrence of auto-antibodies in patients with gastro-duodenal lesions.
AU ***Mardh S.*** ; Song Y.-H.
CS Department of Medical and Physiological Chemistry, Biomedical Centre, Uppsala University, Box 575, S-751 23 Uppsala, Sweden
SO Journal of Internal Medicine, Supplement, (1990) 228/732 (77-82). ISSN: 0955-7873 CODEN: JIMSE3
CY United Kingdom
DT Journal; Conference Article
FS 025 Hematology
026 Immunology, Serology and Transplantation
048 Gastroenterology
LA English
SL English
AB The occurrence of auto-antibodies in patients with the autoimmune disease pernicious anaemia and in patients with active duodenal ulcers was investigated. In order to characterize antigenic structures, various cellular and subcellular fractions were prepared from pig ***gastric*** mucosa and from a homogenate of duodenal mucosa. By means of an enzyme-linked immunosorbent assay and immunoblotting, both the H+,K+-ATPase and pepsinogen/pepsin were shown to constitute the major antigens. All of the seven pernicious-anaemia sera that were tested contained auto-antibodies against both antigens, and the epitopes of the H+,K+-ATPase were shown to be localized on its cytoplasmic face. In 75% (18-24) of the sera from patients with duodenal ulcers, auto-antibodies were detected when using purified antigens. Six sera reacted with H+,K+-ATPase and twelve reacted with pepsinogen, one reacted with both antigens, and four sera reacted with the duodenal mucosal antigen. The occurrence of auto-antibodies indicates that there is a mucosal lesion and that immunological factors may be involved in the pathogenesis of the disease in some patients.

L2 ANSWER 50 OF 86 MEDLINE
AN 90343918 MEDLINE
DN 90343918
TI The occurrence of auto-antibodies in patients with gastro-duodenal lesions.

AU ***Mardh S*** ; Song Y H
CS Department of Medical and Physiological Chemistry, Biomedical Centre, Uppsala University, Sweden.
SO JOURNAL OF INTERNAL MEDICINE. SUPPLEMENT, (1990) 732 77-82. Journal code: ABK. ISSN: 0955-7873.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199011
AB The occurrence of auto-antibodies in patients with the autoimmune disease pernicious anaemia and in patients with active duodenal ulcers was investigated. In order to characterize antigenic structures, various cellular and subcellular fractions were prepared from pig ***gastric*** mucosa and from a homogenate of duodenal mucosa. By means of an enzyme-linked immunosorbent assay and immunoblotting, both the H+,K(+)-ATPase and pepsinogen/pepsin were shown to constitute the major antigens. All of the seven pernicious-anaemia sera that were tested contained auto-antibodies against both antigens, and the epitopes of the H+,K(+)-ATPase were shown to be localized on its cytoplasmic face. In 75% (18/24) of the sera from patients with duodenal ulcers, auto-antibodies were detected when using purified antigens. Six sera reacted with H+,K(+)-ATPase and twelve reacted with pepsinogen, one reacted with both antigens, and four sera reacted with the duodenal mucosal antigen. The occurrence of auto-antibodies indicates that there is a mucosal lesion and that immunological factors may be involved in the pathogenesis of the disease in some patients.

L2 ANSWER 51 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 34
AN 1989:313235 BIOSIS
DN BA88:26965
TI PARIETAL CELL ANTIBODIES IN PERNICIOUS ANEMIA INHIBIT PROTON POTASSIUM ATPASE THE PROTON PUMP OF THE STOMACH.
AU BURMAN P; ***MARDH S*** ; NORBERG L; KARLSSON F A
CS DEP. INTERN. MED., UNIV. HOSP., S-751 85 UPPSALA, SWEDEN.
SO GASTROENTEROLOGY, (1989) 96 (6), 1434-1438. CODEN: GASTAB. ISSN: 0016-5085.
FS BA; OLD
LA English
AB Antibodies to a membrane-bound antigen, localized to the canalicular structures of the parietal cell, are found in most sera of patients with chronic atrophic ***gastritis*** and pernicious anemia. In the present study immunoglobulins containing parietal cell antibodies were found to inhibit the activity of H+,K+-adenosine triphosphatase (EC 3.6.1.36) in a tubulovesicular membrane preparation from porcine ***gastric*** mucosa. The degree of inhibition correlated to the titer of parietal cell antibodies as assessed by an enzyme-linked immunosorbent assay. The specificity of the enzymatic inhibition was confirmed by the lack of effect of parietal cell antibodies on membrane-bound esterase. A possible interaction of parietal cell antibodies with ***gastrin*** binding at the receptor level was investigated in a radioreceptor assay employing 125I- ***gastrin*** 1 and a ***gastric*** mucosal cell suspension from the guinea pig. No blocking capacity was found with immunoglobulins from patients with pernicious anemia as compared with immunoglobulins from healthy controls. The results thus demonstrate a direct inhibitory effect

of parietal cell antibodies on the acid producing H+,K+-adenosine triphosphatase of the parietal cell, but also a lack of interaction with the ***gastrin*** receptor, and indicate that in the development of hypo/achylia H+,K+-adenosine triphosphatase autoantibodies could have a major pathogenic role.

L2 ANSWER 52 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 35
AN 1990:107633 BIOSIS
DN BA89:57124
TI THE EFFECT OF ARACHIDONIC ACID AND ITS METABOLITES ON ACID PRODUCTION IN ISOLATED HUMAN PARIETAL CELLS.
AU JARAMILLO E; ***MARDH S*** ; GREEN K; PERSSON B; RUBIO C; ALY A
CS SECT. GASTROENTEROL., DEP. MED., KAROLINSKA HOSP., S-104 01 STOCKHOLM, SWED.
SO SCAND J GASTROENTEROL, (1989) 24 (10), 1231-1237.
CODEN: SJGRA4. ISSN: 0036-5521.
FS BA; OLD
LA English
AB The effect of arachidonic acid and its metabolites on the histamine-stimulated acid production in human isolated parietal cells provenient from endoscopic biopsies was examined. 14C-aminopyrine (14C-AP) accumulation in the parietal cells was used for evaluation of acid production. Histamine dose-dependently increased AP uptake. Histamine stimulation (taken as 100% at 10-5 M) was significantly inhibited by prostaglandin (PG) E2 to 66 .+-. 7% at 10-8 M, 42 .+-. 8% at 10-6 M, and 13 .+-. 10% at 10-4 M (mean .+-. SEM, n = 10). PGF2.alpha., PGD2, and PGI2 showed significant inhibitory effects only at very high concentrations (10-5-10-4 M). Leukotriene (LT) B4 and LTC4 were without effect. The basal acid production (taken as 0%) was lowered significantly by 10-6 M arachidonic acid to -20 .+-. 7.4% ($p < 0.02$, n = 10), and the histamine-stimulated (10-6 M) acid production from 100% to 64 .+-. 7.2% ($p < 0.001$, n = 10). Aspirin (10-3 M) increased basal (45 .+-. 9.6%, $p < 0.001$, n = 10) and histamine-stimulated (10-6 M) acid production (164 .+-. 16.3%, $p < 0.001$). It is concluded that PGE2, the major product from arachidonic acid metabolism in the human ***gastric*** mucosa, is a significant inhibitor of the histamine-stimulated human parietal cell and may, in humans, play a role as a local physiologic inhibitor of acid secretion.

L2 ANSWER 53 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 36
AN 1989:493408 BIOSIS
DN BA88:119945
TI CHARACTERIZATION OF ANTIGENIC STRUCTURES IN AUTOIMMUNE ATROPHIC ***GASTRITIS*** WITH PERNICIOUS ANEMIA THE PARIETAL CELL PROTON POTASSIUM ATPASE AND THE CHIEF CELL PEPSINOGEN ARE THE TWO MAJOR ANTIGENS.
AU ***MARDH S*** ; SONG Y-H
CS DEP. MED. AND PHYSIOL. CHEM., BIOMEDICAL CENT., UPPSALA UNIV., BOX 575, S-751 23 UPPSALA, SWEDEN.
SO ACTA PHYSIOL SCAND, (1989) 136 (4), 581-588.
CODEN: APSCAX. ISSN: 0001-6772.
FS BA; OLD
LA English
AB Using isolated cells and subcellular fractions from pig ***gastric*** mucosa, antigenic structures with specific binding of IgG from sera of patients with auto-immune atrophic ***gastritis*** were characterized

by means of immunoblotting and enzyme-linked immunosorbent assay. In immunoblotting experiments using mucosal cells as the antigen source, two dominating bands of 94 and 41 kDa were found. The two major antigens were identified as the H,K-ATPase (94 kDa), which constitutes the parietal cell acid pump, and pepsinogen (41 kDa) located in the chief cells. There was also a small but significant binding of antibodies to a preparation of Na,K-ATPase, an enzyme which is about 60% homologous to H,K-ATPase. Commercial preparations of hog ***gastric*** pepsinogen and pepsin bound pernicious anaemia IgG with equal efficacy. When sera from seven patients with the diagnosis pernicious anaemia were tested, all were found to contain auto-against H,K-ATPase as well as pepsinogen. In intact, isolated H,K-ATPase-containing vesicles the cytosolic part of the ATPase molecule is facing the outside of the vesicles. Both intact and trypsinized vesicles were incubated with patient sera and with a monoclonal antibody against H,K-ATPase. Pernicious anaemia IgG was found to bind to a cytosolic, trypsin-resistant structure, but the binding of the monoclonal antibody was lost upon trypsinization. The present results indicate that intracellular structures of the ***gastric*** mucosa, due to cell damage, may be exposed to immune-competent cells, which do not recognize these structures as 'self'.

L2 ANSWER 54 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 37
AN 1990:107457 BIOSIS
DN BA89:56948
TI THE OCCURRENCE OF ***GASTRIC*** AND DUODENAL AUTO-ANTIBODIES IN PEPTIC ULCER DISEASE.
AU SONG Y-H; ***MARDH S***
CS DEP. MED. AND PHYSIOL. CHEM., BIOMEDICAL CENT., UPPSALA UNIV., BOX 575, S-751 23 UPPSALA, SWEDEN.
SO ACTA PHYSIOL SCAND, (1989) 137 (4), 535-540.
CODEN: APSCAX. ISSN: 0001-6772.
FS BA; OLD
LA English
AB The possible relationship between peptic ulcer and the occurrence of auto-antibodies was investigated by means of an enzyme-linked immunosorbent assay (ELISA). Sera from 24 patients with active duodenal ulcer were analysed using cells and subcellular fractions from pig ***gastric*** and dudodenal mucosa for bindingof immunoglobulins. Four sera (17%) reacted with a homogenate from duodenal mucosa. Nine sera (38%) were found to contain auto-antibodies against ***gastric*** mucosal cells. The cell-reactive auto-antibodies were shown to bind preferentially to parietal cells and chief cells. In these cells the antigens were identified as H, K-ATPase and pepsinogen respectively. Six sera were positive against purified H,K-ATPase; 12 sera were positive against pepsinogen, and only one of these sera reacted with both H,K-ATPase and pepsinogen. The results show that auto-antibodies ar formed in a large fraction of patients (18/24; 75%) with peptic ulcer disease. The present study further demonstrates that enrichment of antigenic structures is required for obtaining a satisfactory sensitivity in the assay.

L2 ANSWER 55 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 38
AN 1989:446052 BIOSIS
DN BA88:94324
TI THE EFFECTS OF VARIOUS ***GASTRINS*** ON INTRACELLULAR FREE CALCIUM IN ISOLATED PIG PARIETAL CELLS.

AU CABERO J L; REHFELD J F; ***MARDH S***
CS DEP. MED. PHYSIOL. CHEM., UPPSALA UNIV., BOX 575, S-751 23 UPPSALA, SWEDEN.
SO ACTA PHYSIOL SCAND, (1989) 136 (3), 301-308.
CODEN: APSCAX. ISSN: 0001-6772.
FS BA; OLD
LA English
AB ***Gastrin*** 17 (G17) is a potent stimulant of ***gastric*** acid secretion in vivo. In this study, the effects of G17 and some related on intracellular free Ca2+ in isolated pig parietal cells were studied. Both G17 and the synthetic peptide pentagastrin increased intracellular free Ca2+ in a dose-dependent manner over the concentration range 10-6 to 10-6 M, suggesting a specific action. The EC50 values were 3 .times. 10-8 M for G17 and 8 .times. 10-8 M for pentagastrin. The N-terminal tridecapeptide of G17 [(1-13)G17] did not have any effect on intracellular free Ca2+, nor was it able to inhibit the action of G17. A glycine-extended ***gastrin*** [(5-17)G17-Gly)] elicited a small but significant increase in intracellular free Ca2+ although only at 10-6 M. This increase was approximately 20% of that obtained with a similar concentration of G17. Sequential incubations with (5-17)G17 and G17 showed that both peptides increased the intracellular free Ca2+ through the same mechanisms.

L2 ANSWER 56 OF 86 MEDLINE
AN 89072509 MEDLINE
DN 89072509
TI Effects of some antisecretory drugs on acid production, intracellular free Ca2+, and cyclic AMP production in isolated pig parietal cells.
AU ***Mardh S*** ; Song Y H; Wallmark B
CS Dept. of Medical and Physiological Chemistry, Uppsala University, Sweden..
SO SCANDINAVIAN JOURNAL OF GASTROENTEROLOGY, (1988 Oct) 23 (8) 977-82.
Journal code: UCS. ISSN: 0036-5521.
CY Norway
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198903
AB The effects of some inhibitors of acid secretion were tested on isolated, purified pig parietal cells. The cells were stimulated with 10(-4) M histamine, 10(-5) M carbachol, or 10(-7) M pentagastrin. The H,K-ATPase inhibitors SCH 28080 and omeprazole inhibited both the basal and secretagogue-stimulated acid production, as measured by aminopyrine accumulation, irrespective of the type of stimulator used. The IC50 value was 3-5 x 10(-9) M for SCH 28080 and 1-3 x 10(-8) M for omeprazole. Ranitidine inhibited the histamine-stimulated but not the basal acid production. The IC50 value was 2 x 10(-5) M. Stimulation of acid production with carbachol was blocked by pirenzepine, with an IC50 of 6 x 10(-7) M. Pirenzepine (10(-5) M) specifically blocked the carbachol-stimulated increase in cytosolic free Ca2+ in fura-2-loaded cells but not the increase in cytosolic free Ca2+ induced by histamine or pentagastrin. Ranitidine (10(-4) M) prevented the histamine-induced increase in Ca2+ and was also the only one of the four inhibitors which prevented the histamine-stimulated cAMP formation. SCH 28080 (10(-5) M) significantly potentiated the histamine-stimulated increase in cytosolic free Ca2+ and the formation of cAMP, whereas omeprazole (10(-5) M) was without effect.

L2 ANSWER 57 OF 86 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.DUPLICATE 39
AN 89029721 EMBASE
DN 1989029721
TI Inhibition of H,K-ATPase and Na,K-ATPase by DIDS, a disulphonic stilbene derivative.
AU Vega F.V.; Cabero J.L.; ***Mardh S.***
CS Department of Medical and Physiological Chemistry, Biomedical Centre, Uppsala University, S-751 23 Uppsala, Sweden
SO Acta Physiologica Scandinavica, (1988) 134/4 (543-547).
ISSN: 0001-6772 CODEN: APSCAX
CY Sweden
DT Journal
FS 002 Physiology
048 Gastroenterology
LA English
SL English
AB Disulphonic stilbenes are effective inhibitors of an anion exchanger which is present in the plasma membranes of many cells (Cabantchik et al. 1978). In the present study, the effects of 4,4'-diisothiocyano-2,2'-disulphonic stilbene acid (DIDS) on the transport activity of the hydrochloric acid pump isolated from pig stomach (H,K-ATPase, EC 3.6.1.36) were tested. Half-maximal inhibition of proton transport carried out by the H,K-ATPase in the isolated vesicles was observed at micromolar concentrations of DIDS. The effects of DIDS on the adenosine-triphosphatase and p-nitrophenylphosphatase activities of isolated H,K-ATPase were also studied and compared with those of the kinetically and structurally related Na,K-ATPase (EC 3.6.1.37). Half-maximal inhibition of the enzymatic activities of both enzymes were observed in the micromolar range of DIDS. The lipid bilayer of the ***gastric*** vesicle membrane is highly asymmetric and the original cytosolic side is facing the outside of the vesicle. Since DIDS does not readily cross the membrane, it is most likely that DIDS exerts its inhibitory effects by modifying the transport ATPases on their cytosolic sides.

L2 ANSWER 58 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 40
AN 1988:178288 BIOSIS
DN BA85:90390
TI MAJOR PARIETAL CELL ANTIGEN IN AUTOIMMUNE ***GASTRITIS*** WITH PERNICIOUS ANEMIA IS THE ACID-PRODUCING PROTON POTASSIUM ATPASE OF THE STOMACH.
AU KARLSSON F A; BURMAN P; LOOF L; ***MARDH S***
CS DEP. INTERN. MED., UNIV. HOSP., S-751 85 UPPSALA, SWED.
SO J CLIN INVEST, (1988) 81 (2), 475-479.
CODEN: JCINAO. ISSN: 0021-9738.
FS BA; OLD
LA English
AB In autoimmune ***gastritis*** antibodies against a membrane-bound parietal cell antigen of previously unknown function are present in the sera of patients. In this study, a vesicular membrane preparation of porcine ***gastric*** mucosa cells was found to be a potent antigenic source. This preparation blocked > 90% of antibody binding to a lysate of ***gastric*** mucosa cells. The membrane fraction contained H+,K+-ATPase (EC 3.6.1.36) as the major protein, which in sodium dodecyl sulfate-polyacrylamide gel electrophoresis migrated with a weight of 92

kD. After reduction and alkylation, this component was resolved into two bands of similar staining intensity (92 and 88 kD). Immunoblotting analysis showed that sera of patients recognized antigen with pattern identical to the major protein of the vesicular membranes. Protein A-Sepharose beads preincubated with immunoglobulins of five individual patient (but not control) sera were all found to reduce both the H+,K+-ATPase activity and the amount of parietal cell antigen of a preparation of vesicular membranes solubilized in n-octylglucoside. Taken together, the results of this study indicate that the major parietal cell antigen is identical to the acid-producing enzyme, H+K+-ATPase, of the parietal cell.

L2 ANSWER 59 OF 86 MEDLINE
AN 89128963 MEDLINE
DN 89128963
TI An endogenous inhibitor of Na,K-ATPase isolated from human plasma inhibits the acid pump of the stomach.
AU ***Mardh S***
CS Department of Medical and Physiological Chemistry, Uppsala University, Sweden.
SO PROGRESS IN CLINICAL AND BIOLOGICAL RESEARCH, (1988) 268B 417-22.
Journal code: PZ5. ISSN: 0361-7742.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198905

L2 ANSWER 60 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 41
AN 1988:225334 BIOSIS
DN BA85:114569
TI ADRENALINE STIMULATES ACID PRODUCTION IN ISOLATED PIG AND HUMAN PARIETAL CELLS.
AU SONG Y-H; ***MARDH S*** ; NYREN O; LOOF L
CS DEP. MED. PHYSIOL. CHEM., UPPSALA UNIV. BIOMED. CENT., BOX 575, S-751 23 UPPSALA, SWEDEN.
SO SCAND J GASTROENTEROL, (1988) 23 (1), 35-41.
CODEN: SJGRA4. ISSN: 0036-5521.
FS BA; OLD
LA English
AB To investigate the mechanisms of adrenergic stimulation of the parietal cell and to study the possible relationship between the stress hormone adrenaline and duodenal ulcer, the effects of adrenaline and various adrenoceptor agonists and antagonists were investigated in parietal cells isolated from pig stomachs and from endoscopic biopsy specimens taken from the ***gastric*** mucosa of patients. Parietal cell acid production was assayed by the aminopyrine accumulation technique. Adrenaline as the sole drug showed poor or no stimulatory effect but potentiated histamine-stimulated acid production. In the presence of histamine, beta-adrenoceptor agonists caused a stimulation of acid formation with the potency order isoproterenol>adrenaline>noradrenaline. The beta-2-selective antagonist ICI118551 was a more potent inhibitor of acid production than both the beta-1 antagonist practolol and the H2-receptor antagonist cimetidine. Studies of (3H)-dihydroalprenolol (DHA) binding to purified parietal cell membranes showed a protein-concentration-dependent and

specific binding of 2.2 .+-. 0.6 pmol DHA/.mu.g. Adrenaline increased ***gastric*** acid production in both pig and human parietal cells, most likely through a beta-2 receptor on the parietal cell. The adrenaline stimulatory effect in cells obtained from patients wtih peptic ulcer was more pronounced than in cells from non-ulcer patients, which indicates a possible role of adrenaline in some types of ulcer disease.

L2 ANSWER 61 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 42
AN 1988:133859 BIOSIS
DN BA85:68686
TI ELISA OF PROTON POTASSIUM ATPASE THE PARIETAL CELL ANTIGEN.
AU KARLSSON F A; BURMAN P; LOOF L; OLSSON M; SCHEYNIUS A; ***MARDH S***
CS DEP. INTERN. MED., UNIV. HOSP., S-751 85 UPPSALA, SWED.
SO CLIN EXP IMMUNOL, (1987) 70 (3), 604-610.
CODEN: CEXIAL. ISSN: 0009-9104.
FS BA; OLD
LA English
AB Vesicular membranes, purified from porcine ***gastric*** mucosa and rich in H+, K+-ATPase, were used to establish an enzyme-linked immunosorbent assay (ELISA) for determinations of parietal cell autoantibodies. Results obtained with the ELISA correlated well with standard immunofluorescence determinations of parietal cell antibodies based on frozen sections of rat stomach. The ELISA however was about 10-fold more sensitive than the immunofluorescence method and had high specificity. Intra- and interassay coefficients of variation, determined with a patient sera of average positivity, were 5.5% and 18%, respectively. The ELISA detected antibody binding in 23 out of 26 sera from patients with known autoimmune atrophic ***gastritis*** , in five of 25 sera with autoimmune thyroiditis, in five of 20 sera from patients with Graves' disease, in three out of 20 sera from patients with atoxic nodular goitre, in six of 20 sera of patients with primary biliary cirrhosis, in two out of 20 sera of patients with active duodenal ulcer, in two out of 20 sera with detectable antinuclear antibodies, and in one out of 20 sera with detectable rheumatoid factor. Data determined by an ELISA based on a ***gastric*** vesicular membrane preparation of human origin correlated well ($r = 0.79$, $P < 0.001$) to those obtained by the standard ELISA based on porcine membrane material. The assay should be well suited for routine determinations of parietal cell antibodies in investigations of autoimmune ***gastritis*** and multiple organ autoimmune endocrinopathies.

L2 ANSWER 62 OF 86 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.DUPLICATE 43
AN 88066330 EMBASE
DN 1988066330
TI Mechanisms of stimulation of acid production in parietal cells isolated from the pig ***gastric*** mucosa.
AU ***Mardh S.*** ; Song Y.-H.; Carlsson C.; Bjorkman T.
CS Department of Medical and Physiological Chemistry, Biomedical Centre, Uppsala University, S-751 23 Uppsala, Sweden
SO Acta Physiologica Scandinavica, (1987) 131/4 (589-598).
ISSN: 0001-6772 CODEN: APSCAX
CY Sweden
DT Journal
FS 002 Physiology
029 Clinical Biochemistry

048 Gastroenterology
037 Drug Literature Index
LA English
SL English
AB Sequential incubations with pronase and collagenase of pig ***gastric*** mucosa resulted in single cell preparations containing 10-20% parietal cells, which could be enriched further to 85-95% purity by density-gradient centrifugation followed by elutriation. Acid production of the isolated cells was measured by means of aminopyrine accumulation in their acid compartments. When small pieces of the mucosa were pretreated for 1 h in the presence of either histamine, pentagastrin or carbachol before preparation of cells, the ability of the subsequently isolated cells to produce acid was increased. In parietal cells isolated from resting (not pretreated) mucosa pentagastrin, carbachol and also adrenaline increased the histamine-stimulated aminopyrine accumulation (50-90% increase). Adrenaline alone had no significant effect on the aminopyrine accumulation. In the presence of 10-4 M histamine the apparent EC50 for adrenaline was 5 x 10-7 M. Adrenaline, histamine, forskolin and isobutylmethylxanthin (IBMX) increased the formation of cAMP in purified parietal cells. The three 'classical' secretagogues histamine, pentagastrin and carbachol, but also IBMX and forskolin, increased the cytosolic free Ca2+ from approximately 1.5 x 10-7 M to 2.2 - 3.5 x 10-7 M but adrenaline and dibutyryl cyclic AMP did not. Thus the present results indicate that there are - in addition to histaminergic H2 receptors - specific cholinergic, ***gastrinergic*** and adrenergic receptors on the plasma membrane and that there are separate cAMP and Ca2+-dependent stimulatory pathways in the parietal cell.

L2 ANSWER 63 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1987:435714 BIOSIS
DN BR33:94541
TI ADRENALINE STIMULATES ACID PRODUCTION THROUGH BETA-2 RECEPTORS ON THE PARIETAL CELL.
AU SONG Y; NYREN O; LOOF L; ***MARDH S***
CS DEP. MED., UPPSALA UNIV. BIOMED. CENT., UPPSALA, SWED.
SO TWENTIETH SCANDINAVIAN CONFERENCE ON GASTROENTEROLOGY AND ELEVENTH SCANDINAVIAN MEETING ON GASTROINTESTINAL ENDOSCOPY, TRONDHEIM, NORWAY, JUNE 10-13, 1987. SCAND J GASTROENTEROL SUPPL. (1987) 22 (135), 7. CODEN: SJGSB8. ISSN: 0085-5928.
DT Conference
FS BR; OLD
LA English

L2 ANSWER 64 OF 86 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 86:652689 SCISEARCH
GA The Genuine Article (R) Number: E8786
TI EFFECTS OF PH ON THE ***GASTRIC*** -ACID PRODUCTION MECHANISM
AU VEGA F V (Reprint); ***MARDH S***
CS UNIV NACL MAR DEL PLATA, DEPT BIOL, MAR DEL PLATA, ARGENTINA
CYA ARGENTINA
SO MEDICINA-BUENOS AIRES, (1986) Vol. 46, No. 5, pp. 494-495.
DT Conference; Journal
FS LIFE; CLIN
LA Spanish
REC No References

L2 ANSWER 65 OF 86 MEDLINE
AN 86182986 MEDLINE
DN 86182986
TI Stimulation of acid formation by histamine, carbachol and pentagastrin in isolated pig parietal cells.
AU Norberg L; Ljungstrom M; Vega F V; ***Mardh S***
SO ACTA PHYSIOLOGICA SCANDINAVICA, (1986 Mar) 126 (3) 385-90.
Journal code: 1U4. ISSN: 0001-6772.
CY Sweden
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198607
AB Free cells were obtained by sequential incubations of pig ***gastric*** mucosa with pronase and collagenase. Approximately 10-15% of the cell population represented parietal cells. Accumulation of aminopyrine (AP) in the acid compartments of parietal cells was used as an index of their acid production. Histamine, carbachol and pentagastrin each independently stimulated aminopyrine accumulation. The initial rate of aminopyrine accumulation, observed after addition of 10(-4) M carbachol or 10(-6) M pentagastrin, were 32% and 10%, respectively, of that observed with 10(-4) M histamine. Steady-state aminopyrine accumulation in the presence of 10(-4) M histamine, 10(-4) M carbachol or 10(-6) M pentagastrin were 6.2 +/- 3.3, 2.6 +/- 0.6 and 3.0 +/- 1.5 pmol AP per 10(4) parietal cells, respectively (mean +/- SD, n = 5). The EC50 value for histamine was 3.4 +/- 1.4 X 10(-7) M, and for pentagastrin 5.9 +/- 4.2 X 10(-8) M (mean +/- SD, n = 5). The dose-response curve for carbachol was biphasic. A plateau was reached at 10(-5)-10(-4) M carbachol, and for this phase an apparent EC50 of 2.1 +/- 1.4 X 10(-6) M carbachol was calculated (mean +/- SD, n = 5). A further increase to 10(-3) M carbachol increased the aminopyrine accumulation. Atropine (10(-6) M) inhibited the response to concentrations up to 10(-4) M carbachol, but was without effect on the histamine- and pentagastrin-stimulation. The H2-receptor antagonist, cimetidine, right-shifted the dose--response curve for histamine. Also, the pentagastrin-stimulated aminopyrine accumulation was inhibited by cimetidine.(ABSTRACT TRUNCATED AT 250 WORDS)

L2 ANSWER 66 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 44
AN 1986:91765 BIOSIS
DN BA81:2181
TI PHOSPHOLIPID ORGANIZATION IN PROTON POTASSIUM ATPASE-CONTAINING MEMBRANES FROM PIG ***GASTRIC*** MUCOSA.
AU OLAISSON H; ***MARDH S*** ; ARVIDSON G
CS DEP. MED. AND PHYSIOLOGICAL CHEMISTY, BIOMED. CENTER, UNIV. UPPSALA, BOX 575, S-751 23 UPPSALA, SWEDEN.
SO J BIOL CHEM, (1985) 260 (20), 11262-11267.
CODEN: JBCHA3. ISSN: 0021-9258.
FS BA; OLD
LA English
AB The transverse distribution of the phospholipids in vesicular H+-translocating membranes prepared from pig ***gastric*** mucosa was investigated with the aid of phospholipase C, spingomyelinase, and trinitrobenzenesulfonic acid. The major part (80-90%) of the phosphatidylcholine and the phosphatidylethanolamine, 60% of the

phosphatidylserine, and 45% of the sphingomyelin was located on the external, cytoplasmic side of the vesicle membranes. After treatment with phospholipase C the vesicles still behaved as osmometers and appeared as closed vesicles on the electron micrographs. 31P NMR indicated that the phospholipids in untreated vesicles as well as the unhydrolyzed phospholipids in phospholipase C-treated vesicles were arranged in lamellar structures. The 31P NMR spectrum of untreated vesicles to which Pr3+ ions had been added supported the conclusion that the major part of the membrane phospholipids was located on the external surface of the vesicles. A small fraction of the lipids, 3.6 mol %, was found to consist of glycosphingolipids which occurred at a concentration of 52 nmol/mg of protein.

L2 ANSWER 67 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 45
AN 1985:368521 BIOSIS
DN BA80:38513
TI KINETICS OF THE ACID PUMP IN THE STOMACH PROTON TRANSPORT AND HYDROLYSIS OF ATP AND P NITROPHENYLPHOSPHATE BY THE ***GASTRIC*** PROTON POTASSIUM ATPASE.
AU LJUNGSTROM M; ***MARDH S***
CS DEPARTMENT MEDICAL AND PHYSIOLOGICAL CHEMISTRY, BIOMEDICAL CENTER, BOX 575, S-751 23 UPPSALA, SWEDEN.
SO J BIOL CHEM, (1985) 260 (9), 5440-5444.
CODEN: JBCHA3. ISSN: 0021-9258.
FS BA; OLD
LA English
AB Hydrolysis of ATP and p-nitrophenyl phosphate by pig H,K-ATPase was investigated. Hydrolysis of ATP was studied at pH 7.4 in vesicles treated with the ionophore nigericin. The kinetic analysis showed negative cooperativity with one high affinity (Km1 = 3 .mu.M) and one low affinity (Km2 = 208 .mu.M) site for ATP. The rate of hydrolysis decreased at 2000 .mu.M ATP indicating a 3rd site for ATP. When the pH was decreased to 6.5 the experimental results followed Michaelis-Menten enzyme kinetics with one low affinity site (Km = 116 .mu.M). Higher concentrations than 750 .mu.M ATP were inhibitory. Proton transport was measured as accumulation of acridine orange in vesicles equilibrated with 150 mM KCl. The transport at various concentrations of ATP in the pH interval from 6.0-8.0 correlated well with the Hill equation with a Hill coefficient between 1.5-1.9. The concentration of ATP resulting in half-maximal transport rate (S0.5) increased from 5 .mu.M at pH 6.0 to 420 .mu.M at pH 8.0. At acidic pH the rate of proton transport decreased at 1000 .mu.M ATP. The K+-stimulated p-nitrophenylphosphatase (pNPPase) activity resulted in a Hill coefficient close to 2 indicating cooperative binding of substrate. The pNPPase was noncompetitively inhibited by ATP and ADP; half-maximal inhibition was obtained at 2 and 100 .mu.M, respectively. Phospholipase C-treated vesicles lost 80% of the pNPPase activity, but the Hill coefficient did not change. These kinetic results are used for a further development of the reaction scheme of the H,K-ATPase.

L2 ANSWER 68 OF 86 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 85:505937 SCISEARCH
GA The Genuine Article (R) Number: AQQ34
TI PHOSPHOLIPID ORGANIZATION IN H,K-ATPASE-CONTAINING MEMBRANES FROM PIG ***GASTRIC*** -MUCOSA
AU OLAISSON H (Reprint); ***MARDH S*** ; ARVIDSON G

CS UNIV UPPSALA, CTR BIOMED, DEPT MED & PHYSIOL CHEM, BOX 575, S-75123 UPPSALA, SWEDEN (Reprint)
CYA SWEDEN
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1985) Vol. 260, No. 20, pp. 1262-1267.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 24

L2 ANSWER 69 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 46
AN 1986:145323 BIOSIS
DN BA81:55739
TI OMEPRAZOLE CIMETIDINE AND RANITIDINE INHIBITION OF ACID PRODUCTION IN ISOLATED HUMAN PARIETAL CELLS.
AU GUSTAVSSON S; ***MARDH S*** ; NORBERG L; NYREN O; WOLLERT S
CS DEP. SURG., UNIV. HOSP., S-751 85 UPPSALA, SWED.
SO SCAND J GASTROENTEROL, (1985) 20 (8), 917-921.
CODEN: SJGRA4. ISSN: 0036-5521.
FS BA; OLD
LA English
AB The antisecretory properties of omeprazole, cimetidine, and ranitidine were studied in vitro, using human ***gastric*** mucosal cells, which were obtained by sequential pronase and collagenase incubation of small tissue specimens obtained by endoscopic biopsy. Acid production was measured as the accumulation of radioactive aminopyrine in the acid compartments of the parietal cells. Acid production was stimulated via H2-receptors by histamine (10-4 M or 5 .times. 10-6 M) and via intracellular mechanisms by db-cAMP (10-3 M). Omeprazole induced a dose-dependent inhibition of acid production for all stimulators (IC50 = 2 .times. 10-7 M and 3 .times. 10-8 M with high and low concentrations of histamine, respectively, and 5 .times. 10-6 M with db-cAMP). The H2-receptor antagonists dose-dependently inhibited the histamine-stimulated acid production (IC50 for cimetidine = 10-5 M and 10-6 M and for ranitidine = 10-5 M and 2 .times. 10-7 M for high and low concentrations of histamine, respectively). Neither cimetidine nor ranitidine inhibited acid production after intracellular stimulation with db-cAMP. Omeprazole reduced the aminopyrine accumulation stimulated by histamine (10-4 M) already within 5-10 min, whereas cimetidine (10-3 M) and ranitidine (10-4 M) required 20-30 min. The unstimulated level of acid production was also inhibited by omeprazole but not by the H2-receptor antagonists.

L2 ANSWER 70 OF 86 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.DUPLICATE 47
AN 86082312 EMBASE
DN 1986082312
TI Distribution of carbonic anhydrase in cells and membranes isolated from pig ***gastric*** mucosa.
AU Vega F.V.; Olaisson H.; ***Mardh S.***
CS Departmento de Biologia, Facultad de Ciencias Exactas y Naturales, Universidad Nacional de Mar del Plata, Mar del Plata, Argentina
SO Acta Physiologica Scandinavica, (1985) 124/4 (573-579).
CODEN: APSCAX
CY Sweden
DT Journal
FS 002 Physiology

037 Drug Literature Index
048 Gastroenterology
029 Clinical Biochemistry
LA English
AB Mucosal cells were isolated from pig stomach and then fractionated on linear density gradients of Percoll. Different types of cells were identified by their typical staining and morphology. In disrupted cell fractions, hydration of CO2 by carbonic anhydrase was measured by means of pH-state technique. Localization of carbonic anhydrase to certain cell fractions was also studied by histochemical staining. Both parietal cells and carbonic anhydrase were confined to the low and intermediate density fractions of the gradients. Purified membranes from pig ***gastric*** mucosa, which contained the acid pump of the stomach, the H,K-ATPase, also contained a firmly bound carbonic anhydrase of high activity. The enzyme activity in the membranes was inhibited by acetazolamide, furosemide and KSCN. The molecular mass of the carbonic anhydrase was 33 kDA as estimated by its binding of [14C]furosemide followed by polyacrylamide gel electrophoresis. Previous suggestions of a role of carbonic anhydrase as a supplier of H+ in the secretion of acid are supported by its high activity of its localization to the same membrane as the acid pump of the stomach.

L2 ANSWER 71 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 48
AN 1985:332156 BIOSIS
DN BA80:2148
TI A METHOD FOR IN-VITRO STUDIES ON ACID FORMATION IN HUMAN PARIETAL CELLS STIMULATION BY HISTAMINE PENTAGASTRIN AND CARBACHOL.
AU ***MARDH S*** ; NORBERG L; JUNGSTROM M L; WOLLERT S; NYREN O; GUSTAVSSON S
CS DEP. MED. PHYSIOL. CHEM., BIOMED. CENT., UPPSALA UNIV., UPPSALA, SWEDEN.
SO ACTA PHYSIOL SCAND, (1985) 123 (3), 349-354.
CODEN: APSCAX. ISSN: 0001-6772.
FS BA; OLD
LA English
AB Cells were isolated from human ***gastric*** mucosa on a large scale from ***gastric*** resection specimens and on a microscale from endoscopic biopsies by sequential incubations with pronase and collagenase. The accumulation of aminopyrine (AP) was used as an index of acid production in the parietal cells. Basal accumulation was about 0.2 pmol AP/104 parietal cells. Addition of histamine, db [dibutyl]-cAMP, pentagastrin and carbachol increased the aminopyrine accumulation. Maximal accumulation was of the order of 1000-2800% of the control and was obtained after stimulation by 10-4 M histamine and by 10-3 M db-cAMP. Stimulation by pentagastrin and by carbachol reached 200 to 350% of the control. EC50 was 2 x 10-6 M for histamine, 10-8 M for pentagastrin and 4 x 10-6 M for carbachol. Human parietal cells were enriched from a mixture of ***gastric*** mucosal cells by isopycnic centrifugation on density gradients of Percoll. A parietal cell fraction with a purity of 83% was obtained. The density of human parietal cells was estimated to 1.06 g/ml.

L2 ANSWER 72 OF 86 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 85:504085 SCISEARCH
GA The Genuine Article (R) Number: AQB33
TI PRELIMINARY CHARACTERIZATION OF LOW-DENSITY PLASMA-MEMBRANE FROM BOVINE ***GASTRIC*** -MUCOSA RELATED WITH THE MECHANISM OF ***GASTRIC*** -ACID SECRETION

AU VEGA F V (Reprint); RODRIGUEZ M P; ***MARDH S***
CS UNIV NACL MAR DEL PLATA, FAC CIENCIAS EXACTAS & NAT, DEPT BIOL, RA-7600 MAR DEL PLATA, ARGENTINA; UNIV UPPSALA, CTR BIOMED, DEPT MED & PHYSIOL CHEM, S-75123 UPPSALA, SWEDEN
CYA ARGENTINA; SWEDEN
SO ACTA PHYSIOLOGICA SCANDINAVICA, (1985) Vol. 124, pp. 123.
DT Conference; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 4

L2 ANSWER 73 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 49
AN 1984:133748 BIOSIS
DN BR27:50240
TI PURIFICATION OF PARIETAL CELLS FROM HUMAN ***GASTRIC*** MUCOSA BY CENTRIFUGATION ON A DENSITY GRADIENT OF PERCOLL.
AU ***MARDH S*** ; NORBERG L; LJUNGSTROM M; WOLLERT S; ADAMI H-O; NYREN O; LOOF L; GUSTAVSSON S
CS DEPARTMENT OF MEDICAL AND PHYSIOL CHEM., UNIVERSITY OF UPPSALA, SWEDEN.
SO THE 85TH ANNUAL MEETING OF THE AMERICAN GASTROENTEROLOGICAL ASSOCIATION HELD IN CONJUNCTION WITH THE AMERICAN ASSOCIATION FOR THE STUDY OF LIVER DISEASE, AND THE GASTROENTEROLOGY STUDY GROUP, NEW ORLEANS, LA., USA, MAY 19-25, 1984. GASTROENTEROLOGY. (1984) 86 (5 PART 2), 1173.
CODEN: GASTAB. ISSN: 0016-5085.
DT Conference
FS BR; OLD
LA English

L2 ANSWER 74 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 50
AN 1985:322910 BIOSIS
DN BA79:102906
TI PREPARATION OF CELLS FROM PIG ***GASTRIC*** MUCOSA ISOLATION OF PARIETAL CELLS BY ISOPYCNIC CENTRIFUGATION ON LINEAR DENSITY GRADIENTS OF PERCOLL.
AU ***MARDH S*** ; NORBERG L; LJUNGSTROM M; HUMBLE L; BORG T; CARLSSON C
CS DEP. MED. PHYSIOL. CHEM., BIOMED. CENT., UPPSALA UNIV., BOX 575, S-75123 UPPSALA, SWED.
SO ACTA PHYSIOL SCAND, (1984 (RECD 1985)) 122 (4), 607-614.
CODEN: APSCAX. ISSN: 0001-6772.
FS BA; OLD
LA English
AB Cells were isolated from pig ***gastric*** mucosa by a combination of mechanical and enzymatic treatment. Isopycnic centrifugation on linear density gradients of Percoll provided a simple and rapid procedure for obtaining highly enriched parietal cells and chief cells. Their densities were 1.06 and 1.10 g/ml, respectively. Isolated mucosal cells attached to Petri dishes coated with fibronectin or collagen. Both parietal cells and chief cells adhered more readily to fibronectin than collagen. Mucosal cells and cells from the Percoll gradients were maintained for up to 1 wk as primary cell cultures. The ability of free parietal cells to produce acid was tested by the 14C-aminopyrine accumulation technique. Maximal accumulation was 2.5 pmol aminopyrine/104 parietal cells after incubation for 45 min at 10-4 M histamine. The EC50 [median effective concentration] for histamine was 5 .times. 10-6 M. The accumulation of aminopyrine at 10-6 M carbachol and 10-7 M pentagastrin were for both secretagogues about

0.9 pmol per 104 parietal cells.

L2 ANSWER 75 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 51
AN 1984:306266 BIOSIS
DN BA78:42746
TI EFFECTS OF PH ON THE INTERACTION OF LIGANDS WITH THE PROTON POTASSIUM ION ATPASE PURIFIED FROM PIG ***GASTRIC*** MUCOSA.
AU LJUNGSTROM M; VEGA F V; ***MARDH S***
CS DEP. MED. PHYSIOL. CHEM., BIOMEDICAL CENT., UPPSAL UNIV., BOX 575, S-751 23 UPPSALA, SWED.
SO BIOCHIM BIOPHYS ACTA, (1984) 769 (1), 220-230.
CODEN: BBACAQ. ISSN: 0006-3002.
FS BA; OLD
LA English
AB The effects of K+, Na+ and ATP on the ***gastric*** (H+ + K+)-ATPase were investigated at various pH. The enzyme was phosphorylated by ATP with pseudo-1st-order rate constant of 3650 min-1 at pH 7.4. This rate constant increased to a maximal value of .apprx. 7900 min-1 when pH was decreased to 6.0. Alkalinization decreased the rate constant. At pH 8.0 it was 1290 min-1. Additions of 5 mM K+ or Na+, did not change the rate constant at acidic pH, while a neutral or alkaline pH a decrease was observed. Dephosphorylation of phosphoenzyme in lyophilized vesicles was dependent on K+, but not on Na+. Alkaline pH increased the rate of dephosphorylation. K+ stimulated the ATPase and p-nitrophenylphosphatase (pNPPase) activities. At high concentrations K+ was inhibitory. Below pH 7.0 Na+ had little or no effect on the ATPase and p-nitrophenylphosphatase, while at alkaline pH, Na+ inhibited both activities. The effect of extravesicular pH on transport of H+ was investigated. At pH 6.5 the apparent Km for ATP was 2.7 .mu.M and increased little when K+ was added extravesicularly. At pH 7.5, millimolar concentrations of K+ increased the apparent Km for ATP. Extravesicular K+ and Na+ inhibited the transport of H+. The inhibition was strongest at alkaline pH and only slight at neutral or acidic pH, suggesting a competition between the alkali metal ions and H+ at a common binding site on the cytoplasmic side of the membrane. Two H+-producing reactions as possible candidates as physiological regulators of (H+ + K+)-ATPase were investigated. Firstly, the hydrolysis of ATP per se, and secondly, the hydration of CO2 and the subsequent formation of H+ and HCO3-. The amount of H+ formed in the ATPase reaction was highest at alkaline pH. The H+/ATP ratio was .apprx. 1 at pH 8.0. When CO2 was added to the reaction medium there was no change in the rate of H+ transport at pH 7.0, but at pH 8.0 the rate increased 4-times upon the addition of 0.4 mM CO2. A possible cooperation is indicated in the production of acid between H+ + K+-ATPase and a carbonic anhydrase associated with the vesicular membrane is indicated.

L2 ANSWER 76 OF 86 LIFESCI COPYRIGHT 2001 CSA
AN 84:46219 LIFESCI
TI Effects of pH on the interaction of ligands with the (H super(+) + K super(+))-ATPase purified from pig ***gastric*** mucosa.
AU Ljungstroem, M.; Vega, F.V.; ***Mardh, S.***
CS Dep. Med. and Physiol. Chem., Biomed. Cent., Uppsala Univ., Box 575, S-751 23 Uppsala, Sweden
SO BIOCHIM. BIOPHYS. ACTA., (1984) vol. 769, no. 1, pp. 220-230.
DT Journal

FS M
LA English
SL English
AB The effects of K super(+), Na super(+) and ATP on the ***gastric*** (H super(+) + K super(+))-ATPase were investigated at various pH. The enzyme was phosphorylated by ATP at pH 7.4. This increased to a maximal when pH was decreased to 6.0. Alkalinization decreased the rate constant. Dephosphorylation of phosphoenzyme in lyophilized vesicles was dependent on K super(+), but not on Na super(+). Alkaline pH increased the rate of dephosphorylation. K super(+) stimulated the ATPase and p-nitrophenylphosphatase activities. The effect of extravesicular pH on transport of H super(+) was investigated. Extravesicular K super(+) and Na super(+) inhibited the transport of H super(+). The inhibition was strongest at alkaline pH and only slight at neutral or acidic pH, suggesting a competition between the alkali metal ions and hydrogen ions at a common binding site on the cytoplasmic side of the membrane. Two H super(+)-producing reactions as possible candidates as physiological regulators of (H super(+) + K super(+))-ATPase were investigated. The results indicate a possible co-operation in the production of acid between the H super(+) + K super(+)-ATPase and a carbonic anhydrase associated with the vesicular membrane.

L2 ANSWER 77 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 52
AN 1984:305455 BIOSIS
DN BA78:41935
TI CHARACTERIZATION OF PROTON TRANSPORTING MEMBRANES FROM RESTING PIG ***GASTRIC*** MUCOSA.
AU LJUNGSTROM M; NORBERG L; OLAISSON H; WERNSTEDT C; VEGA F V; ARVIDSON G; ***MARDH S***
CS DEP. MED. PHYSIOL. CHEM., BIOMED. CENT., UPPSALA UNIV., BOX 575, S-751 23 UPPSALA, SWED.
SO BIOCHIM BIOPHYS ACTA, (1984) 769 (1), 209-219.
CODEN: BBACAQ. ISSN: 0006-3002.
FS BA; OLD
LA English
AB Membrane vesicles were purified from resting corpus mucosa of pig stomachs by velocity-sedimentation on a sucrose-Ficoll step gradient. Two vesicular fractions containing the (H+ + K+)-ATPase were obtained. One fraction was tight towards KCl, the other was leaky. At 21.degree. C maximal (H+ + K+)-ATPase activities of 0.8 and 0.4 .mu.mol .cntdot. mg-1 .cntdot. min-1, respectively, were observed in lyophilized vesicles. The vesicles contained a membrane-associated carbonic anhydrase, the activity of which was in 100-fold excess of the maximal ATPase activity. Both vesicular fractions were rich in phosphatidylcholine, phosphatidylethanolamine, sphingomyelin and cholesterol. The characteristics of ion permeability and transport in the tight vesicles were in agreement with corresponding data for vesicles of a tubulovesicular origin in the parietal cell. Measurement of the rate of K+ uptake into the vesicles was based on the ability of K+ to promote H+ transport. The uptake was slow and dependent on the type of anion present. The effectiveness in promoting uptake of K+ by anions was SCN- > NO3- > Cl- .mchgt. HCO3- > SO42-. Uptake of K+ was much more rapid at alkaline pH than at neutral or at acidic pH. Addition of CO2 at alkaline pH strongly stimulated the rate of H+ accumulation in the vesicles. The initial part of this stimulation was sensitive to acetazolamide, an inhibitor of carbonic anhydrase. A model how the (H+ +

K+)-ATPase and the carbonic anhydrase may cooperate is presented. Membrane vesicles of a tubulovesicular origin can produce acid.

L2 ANSWER 78 OF 86 LIFESCI COPYRIGHT 2001 CSA
AN 84:46215 LIFESCI
TI Characterization of proton-transporting membranes from resting pig ***gastric*** mucosa.
AU Ljungstroem, M.; Norberg, L.; Olaisson, H.; Wernstedt, C.; Vega, F.V.; Arvidson, G.; ***Mardh, S.***
CS Dep. Med. Physiol. Chem., Biomed. Cent., Uppsala University, Box 575, S-751 23 Uppsala, Sweden
SO BIOCHIM. BIOPHYS. ACTA., (1984) vol. 769, no. 1, pp. 209-219.
DT Journal
FS M
LA English
SL English
AB Membrane vesicles were purified from resting corpus mucosa of pig stomachs by velocity-sedimentation on a sucrose-Ficoll step gradient. Two vesicular fractions containing the (H super(+) + K super(+))-ATPase were obtained. One fraction was tight towards KCl, the other was leaky. The vesicles contained a membrane-associated carbonic anhydrase, the activity of which was in 100-fold excess of the maximal ATPase activity. Both vesicular fractions were rich in phosphatidylcholine, phosphatidylethanolamine, sphingomyelin and cholesterol. The characteristics of ion permeability and transport in the tight vesicles were in agreement with corresponding data for vesicles of a tubulovesicular origin in the parietal cell. Measurement of the rate of K super(+) uptake into the vesicles was based on the ability of K super(+) to promote H super(+) transport. The uptake was slow and dependent on the type of anion present. A model how the (H super(+) + K super(+))-ATPase and the carbonic anhydrase may co-operate is presented. It is concluded that membrane vesicles of a tubulovesicular origin can produce acid.

L2 ANSWER 79 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 53
AN 1983:236534 BIOSIS
DN BA75:86534
TI INHIBITION OF ***GASTRIC*** HYDROGEN POTASSIUM ATPASE BY THE SUBSTITUTED BENZIMIDAZOLE PICOPRAZOLE.
AU WALLMARK B; SACHS G; ***MARDH S*** ; FELLENIUS E
CS AB HASSLE, RES. LAB., S-431 83 MOLNDAL.
SO BIOCHIM BIOPHYS ACTA, (1983) 728 (1), 31-38.
CODEN: BBACAQ. ISSN: 0006-3002.
FS BA; OLD
LA English
AB The substituted benzimidazole, picoprazole, inhibited the [hog] ***gastric*** (H+ + K+)-ATPase in a concentration- and time-dependent manner. Half-maximal inhibition of the (H+ + K+)-ATPase activity was obtained at .apprx. 2 .cntdot. 10-6 M under standard conditions. In addition to the inhibition of ATPase activity, parallel inhibition of phosphoenzyme formation and the proton transport activity were achieved. Radiolabeled picoprazole was bound to a 100 kDa [kilodalton] peptide; this peptide was shown by phosphorylation experiments to contain the catalytic center of the (H+ + K+)-ATPase. Studies on the (Na+ + K+)-ATPase indicated that this enzyme was unaffected by picoprazole. From the data presented and from other pharmacological studies, it is proposed that this compound

inhibits acid secretion at the level of the parietal cell by its ability to inhibit the ***gastric*** proton pump, the (H+ + K+)-ATPase.

L2 ANSWER 80 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 54
AN 1984:16005 BIOSIS
DN BR26:16005
TI EFFECTS OF PHOSPHO LIPASE C ON ***GASTRIC*** VESICLE MEMBRANES CONTAINING HYDROGEN ION POTASSIUM ION ATPASE.
AU OLAISSON H; ***MARDH S*** ; ARVIDSON G
CS INST. OF MED. AND PHYSIOL. CHEM., BIOMED. CENT., UNIV. OF UPPSALA, BOX 575, S-75123 UPPSALA, SWED.
SO Acta Chem. Scand., Ser. B, (1982 (RECD 1983)) 36 (9), 649-650.
CODEN: ACBOCV. ISSN: 0302-4369.
FS BR; OLD
LA English

L2 ANSWER 81 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 55
AN 1983:183275 BIOSIS
DN BA75:33275
TI ATP ADP EXCHANGE ACTIVITY OF ***GASTRIC*** PROTON POTASSIUM ATPASE.
AU RABON E; SACHS G; ***MARDH S*** ; WALLMARK B
CS LAB. MEMBRANE BIOL., UNIV. ALABAMA BIRMINGHAM, BIRMINGHAM, ALA. 35294.
SO BIOCHIM BIOPHYS ACTA, (1982) 688 (2), 515-524.
CODEN: BBACAQ. ISSN: 0006-3002.
FS BA; OLD
LA English
AB . The ATP/ADP exchange is shown to be a partial reaction of the [hog] (H+ + K+)-ATPase by the absence of measurable nucleoside diphosphokinase activity and the insensitivity of the reaction to P1,P5-di(adenosine-5') pentaphosphate, a myokinase inhibitor. The exchange demonstrates an absolute requirement for Mg2+ and is optimal at an ADP/ATP ratio of 2. The high ATP concentration (K0.5 = 116 .mu.M) required for maximal exchange is interpreted as evidence for the involvement of a low affinity form of nucleotide site. The ATP/ADP exchange is regarded as evidence for an ADP-sensitive form of the phosphoenzyme. In native enzyme, pre-steady state kinetics show that the formation of the phosphoenzyme is partially sensitive to ADP while modification of the enzyme by pretreatment with 5,5'-dithiobis(2-nitrobenzoic acid) (DTNB) in the absence of Mg2+ results in a steady-state phosphoenzyme population, a component of which is ADP sensitive. The ATP/ADP exchange reaction can be either stimulated or inhibited by the presence of K+ as a function of pH and Mg2+.

L2 ANSWER 82 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1982:106645 BIOSIS
DN BR23:36637
TI MONOVALENT CATION DEPENDENT REGULATION OF HYDROGEN ION POTASSIUM ATPASE FROM PIG ***GASTRIC*** MUCOSA.
AU LJUNGSTROM M; ***MARDH S***
CS INST. MED. PHYSIOL. CHEM., UPPSALA, SWED.
SO MEETING OF THE BIOCHEMICAL SOCIETY, MARCH 29-APRIL 3, 1981. BIOCHEM SOC TRANS. (1981) 9 (2), 179P.
CODEN: BCSTB5. ISSN: 0300-5127.
DT Conference
FS BR; OLD
LA English

L2 ANSWER 83 OF 86 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 82:635282 SCISEARCH
GA The Genuine Article (R) Number: US435
TI MONO-VALENT CATION-DEPENDENT REGULATION OF H&,K&-ATPASE FROM PIG ***GASTRIC*** -MUCOSA
AU LJUNGSTROM M (Reprint); ***MARDH S***
CS UNIV UPPSALA, INST MED & PHYSIOL CHEM, S-75105 UPPSALA, SWEDEN
CYA SWEDEN
SO BIOCHEMICAL SOCIETY TRANSACTIONS, (1981) Vol. 9, No. 2, pp. P179.
DT Conference; Journal
FS LIFE
LA ENGLISH
REC No References

L2 ANSWER 84 OF 86 MEDLINE
AN 80049837 MEDLINE
DN 80049837
TI Phosphorylation and dephosphorylation kinetics of potassium-stimulated ATP phosphohydrolase from hog ***gastric*** mucosa.
AU Wallmark B; ***Mardh S***
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1979 Dec 10) 254 (23) 11899-902.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198003
AB Partial reactions of potassium-stimulated ATP phosphohydrolase from hog ***gastric*** mucosa were studied by means of a rapid-mixing apparatus. At 21 degrees C, in the presence of 2 mM MgCl2 and 5 microM [gamma-32P]ATP there was a rapid phosphorylation of the enzyme with a pseudofirst order rate constant of 1400 min-1. Addition of the ATP about 120 ms before the MgCl2 increased this rate constant to 4400 min-1. In the absence of MgCl2 there was no phosphorylation. Addition of 4 or 10 mM KCl to the phosphoenzyme which had been formed in the absence of KCl produced a rapid initial rate of dephosphorylation (k = 2600 and 3200 min-1 respectively). An additional slow component of dephosphorylation was observed when unlabeled ATP was added together with the KCl (k = 700 to 900 min-1). At a 4 mM concentration, KCl stimulated the ATPase activity about 9-fold. At higher concentrations, the activity was reduced in parallel with a reduction of the steady state level of phosphoenzyme. Addition of KCl to the enzyme before the addition of ATP plus MgCl2 resulted in a low rate and extent of phosphorylation. KCl appeared to inhibit the phosphorylation at a level preceeding the E.ATP complex.

L2 ANSWER 85 OF 86 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 79:537048 SCISEARCH
GA The Genuine Article (R) Number: HX412
TI PHOSPHORYLATION AND DEPHOSPHORYLATION KINETICS OF POTASSIUM-STIMULATED ATP PHOSPHOHYDROLASE FROM HOG ***GASTRIC*** -MUCOSA
AU WALLMARK B; ***MARDH S (Reprint)***
CS AB HASSLE, DEPT ANALYT CHEM & BIOCHEM, S-43120 MOLNDAL 1, SWEDEN; UNIV UPPSALA, CTR BIOMED, INST MED & PHYSIOL CHEM, S-75123 UPPSALA, SWEDEN
CYA SWEDEN

SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1979) Vol. 254, No. 23, pp. 1899-1902.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 15

L2 ANSWER 86 OF 86 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 56
AN 1980:54306 BIOSIS
DN BR18:54306
TI REGULATION OF INTERMEDIARY PHOSPHORYLATION OF POTASSIUM ATPASE FROM PIG ***GASTRIC*** MUCOSA BY SODIUM IONS.
AU LJUNGSTROM M; WALLMARK B; ***MARDH S***
CS BIOMED. CENT., INST. MED. PHYSIOL. CHEM., UPPS. UNIV., BOX 575, S-751 23 UPPSALA, SWED.
SO Acta Chem. Scand., Ser. B, (1979 (RECD 1980)) 33 (8), 618-619.
CODEN: ACBOCV. ISSN: 0302-4369.
DT Short Communication
FS BR; OLD
LA English

=> e mardh erik/au

E1 3 MARDH C K/AU
E2 2 MARDH CARINA KARRMAN/AU
E3 0 --> MARDH ERIK/AU
E4 61 MARDH G/AU
E5 3 MARDH GORAN/AU
E6 6 MARDH P/AU
E7 1393 MARDH P A/AU
E8 1 MARDH P A */AU
E9 2 MARDH P N/AU
E10 9 MARDH PER A/AU
E11 63 MARDH PER ANDERS/AU
E12 1 MARDH PER ANDERSD/AU

=> s pylori and gastri?

L3 41957 PYLORI AND GASTRI?

=> s pepsinogen and gastri?

L4 5756 PEPSINOGEN AND GASTRI?

=> s atpase and gastri?

L5 8244 ATPASE AND GASTRI?

=> s l3 and l4 and l5

L6 7 L3 AND L4 AND L5

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L7 5 DUP REM L6 (2 DUPLICATES REMOVED)

=> d bib ab 1-

YOU HAVE REQUESTED DATA FROM 5 ANSWERS - CONTINUE? Y/(N):y

L7 ANSWER 1 OF 5 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.DUPLICATE 1
AN 1998239732 EMBASE
TI Serum antibodies to H+,K+- ***ATPase*** , serum ***pepsinogen*** A and Helicobacter ***pylori*** in relation to ***gastric*** mucosa morphology in patients with low or low-normal concentrations of serum cobalamins.
AU Lindgren A.; Burman P.; Kilander A.F.; Nilsson O.; Lindstedt G.
CS Dr. A. Lindgren, Department of Internal Medicine, Boras Central Hospital, S-501 82 Boras, Sweden
SO European Journal of Gastroenterology and Hepatology, (1998) 10/7 (583-588).
Refs: 41
ISSN: 0954-691X CODEN: EJGHES
CY United Kingdom
DT Journal; Article
FS 029 Clinical Biochemistry
048 Gastroenterology
LA English
SL English
AB Objectives. To compare the diagnostic performance of serum antibodies to H+,K+- ***ATPase*** (EC 3.6.1.36), serum ***pepsinogen*** A (EC 3.4.23.1) and the Schilling test in diagnosing chronic atrophic body ***gastritis*** ; to study the interrelationships between H+,K+- ***ATPase*** antibodies, serology for Helicobacter ***pylori*** , and ***gastric*** morphology. Design. Patients with suspected cobalamin deficiency and serum cobalamin < 200 .mu.mol/l were investigated using upper gastrointestinal endoscopy, the Schilling test and serum tests for H+,K+- ***ATPase*** antibodies, ***pepsinogen*** A, and H. ***pylori*** . Setting. The Department of Internal Medicine, Sahlgrenska University Hospital, Goteborg, Sweden. Patients. Ninety seven consecutively referred patients. Main outcome measures. Sensitivity and specificity of assays for serum H+,K+- ***ATPase*** antibodies, serum ***pepsinogen*** A, and the Schilling test. Results. Assays of serum antibodies to H+,K+- ***ATPase*** and of serum ***pepsinogen*** A displayed equal diagnostic sensitivity for atrophic ***gastritis*** (around 0.90 for the severe forms) and higher than that for the Schilling test (0.65). The diagnostic specificity for ***pepsinogen*** A (1.0) was higher than for H+,K+- ***ATPase*** antibodies (about 0.80). The prevalence of antral ***gastritis*** and positivity for H. ***pylori*** antibodies declined with the transition of body ***gastritis*** into severe atrophy, while the prevalence of H+,K+- ***ATPase*** antibodies increased. Conclusion. ***Pepsinogen*** A is preferable to serum H+,K+- ***ATPase*** antibodies in the diagnosis of ***gastric*** body mucosal atrophy. The formation of H+,K+- ***ATPase*** antibodies does not seem to be a primary event in the development of ***gastric*** body muscosal atrophy.

L7 ANSWER 2 OF 5 SCISEARCH COPYRIGHT 2001 ISI (R)

AN 97:876684 SCISEARCH
GA The Genuine Article (R) Number: YG753
TI Regulation of ***gastric*** secretion
AU Schubert M L (Reprint)
CS MCGUIRE DEPT VET AFFAIRS MED CTR, DIV GASTROENTEROL, CODE 111N, 1201 BROAD ROCK BLVD, RICHMOND, VA 23249 (Reprint)
CYA USA
SO CURRENT OPINION IN GASTROENTEROLOGY, (NOV 1997) Vol. 13, No. 6, pp. 441-450.
Publisher: RAPID SCIENCE PUBLISHERS, 2-6 BOUNDARY ROW, LONDON, ENGLAND SE1 8NH.
ISSN: 0267-1379.
DT Article; Journal
FS CLIN
LA English
REC Reference Count: 83
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB ***Gastric*** secretion is finely regulated by neural, hormonal, and paracrine pathways, During ingestion of a meal, the pathways can be activated by stimuli originating in the brain or stimuli originating in the stomach, such as mechanical stimulation leg, distension) or chemical stimulation leg, protein). The main secretagogues active at the level of the parietal cell are acetylcholine (neurotransmitter), ***gastrin*** (hormone), and histamine (paracrine agent); the main inhibitor is somatostatin (paracrine agent). The release of these four agents by neural, hormonal, and paracrine mechanisms and the interactions among them determine the rate of acid secretion in response to physiological stimuli. The two main intracellular signaling pathways involve cyclic adenosine monophosphate and calcium. A third pathway, involving accumulation of guanosine 3',5'-cyclic monophosphate, is receiving increased attention, Progress has been made in characterizing the intracellular events that occur between generation of second messengers and incorporation of tubulovesicle-rich H+K+- ***ATPase*** into the apical membrane of the parietal cell and exocytosis of ***pepsinogen*** from the chief cell.

L7 ANSWER 3 OF 5 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1998:25516 BIOSIS
DN PREV199800025516
TI Helicobacter ***pylori*** associated autoantibodies recognize Lewis antigens, and peptide epitopes of ***gastric*** H+, K+- ***ATPase*** and intrinsic factor.
AU Appelmelk, B. J. (1); Straver, S. (1); Claeys, D.; Faller, G.; Kirchner, T.; Negrini, R.; Krakowka, S.; Eaton, K.; Vandenbroucke-Grauls, C. M. J. E. (1)
CS (1) Vrije Univ., Amsterdam Netherlands
SO Gut, (1997) Vol. 41, No. SUPPL. 1, pp. A17.
Meeting Info.: European Helicobacter Pylori Study Group Xth International Workshop on Gastroduodenal Pathology and Helicobacter Pylori Lisbon, Portugal September 11-14, 1997 European Helicobacter pylori Study Group . ISSN: 0017-5749.
DT Conference
LA English

L7 ANSWER 4 OF 5 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 94:715221 SCISEARCH

GA The Genuine Article (R) Number: PQ251
TI POSITIVE CORRELATION BETWEEN H,K-ADENOSINE TRIPHOSPHATASE AUTOANTIBODIES AND HELICOBACTER- ***PYLORI*** ANTIBODIES IN PATIENTS WITH PERNICIOUS-ANEMIA
AU MA J Y; BORCH K; SJOSTRAND S E; JANZON L; MARDH S (Reprint)
CS LINKOPING UNIV, FAC HLTH SCI, DEPT CELL BIOL, S-58185 LINKOPING, SWEDEN (Reprint); LINKOPING UNIV, FAC HLTH SCI, DEPT CELL BIOL, S-58185 LINKOPING, SWEDEN; LINKOPING UNIV HOSP, DEPT MEDICOSURG GASTROENTEROL, S-58185 LINKOPING, SWEDEN; UPPSALA UNIV, CTR BIOMED, DEPT MED & PHYSIOL CHEM, UPPSALA, SWEDEN; AB ASTRA & AB ASTRA ARCUS, SODERTALJE, SWEDEN
CYA SWEDEN
SO SCANDINAVIAN JOURNAL OF GASTROENTEROLOGY, (NOV 1994) Vol. 29, No. 11, pp. 961-965.
ISSN: 0036-5521.
DT Article; Journal
FS LIFE; CLIN
LA ENGLISH
REC Reference Count: 30
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Background: Helicobacter ***pylori*** is a major cause of ***gastritis*** , and the parietal cell H,K-adenosine triphosphatase (***ATPase***) is a major autoantigen in autoimmune atrophic corpus ***gastritis*** , which may eventually lead to pernicious anemia and/or neuropathy. Whether the bacterium induces the autoimmune response is unknown. Methods: By means of enzyme-linked immunosorbent assay the occurrence of antibodies against porcine H,K- ***ATPase*** and H. ***pylori*** was determined in sera from 30 patients with pernicious anemia. Results: All sera scored positive against H,K- ***ATPase*** , and 25 (83%) scored positive against H. ***pylori*** . The titers of antibodies against both antigen preparations inversely correlated with the duration of disease. A possible common epitope in the antigen preparations was tested with a competition assay. There was no indication of a common epitope in either human or porcine H,K- ***ATPase*** and H. ***pylori*** . Conclusions: There was a positive correlation and a high incidence of antibodies against H,K- ***ATPase*** and H. ***pylori*** in sera from patients with pernicious anemia. These antibodies recognized different epitopes.

L7 ANSWER 5 OF 5 MEDLINE
AN 91047803 MEDLINE
DN 91047803
TI Acid and barriers. Current research and future developments for peptic ulcer therapy.
AU Rademaker J W; Hunt R H
CS Division of Gastroenterology, McMaster University Medical Centre, Hamilton, Ontario, Canada..
SO SCANDINAVIAN JOURNAL OF GASTROENTEROLOGY. SUPPLEMENT, (1990) 175 19-26.
Ref: 67
Journal code: UCT. ISSN: 0085-5928.
CY Norway
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals

EM 199102
AB Medical therapy for peptic ulcer disease has been targeted at inhibiting acid secretion based on the belief that ulcers occur due to an imbalance between aggressive and protective factors. New antisecretory agents are unlikely to show any dramatic improvement over the success and safety of histamine H2 receptor antagonists or the recently introduced H+K+ ***ATPase*** proton pump antagonist omeprazole. The development of specific muscarinic M3 and ***gastrin*** receptor antagonists will provide useful agents to suppress acid and ***pepsinogen*** secretion by alternative means and may prevent the associated hypergastrinaemia seen with anti-secretory therapy. Enhancement of mucosal defence by site protective agents will be based on a better understanding of the vascular and immune factors involved in maintaining mucosal integrity and the growth factors that regulate wound healing. Molecular techniques are likely to produce the 'model anti-ulcer' agent which will effectively inhibit acid secretion and also enhance wound healing thus providing a cure for this chronic disease.

=> s antibod? and (l3 or l4 or l5)

L8 7055 ANTIBOD? AND (L3 OR L4 OR L5)

=> s l8 and (immunoassaY?)

L9 564 L8 AND (IMMUNOASSAY?)

=> dup rem l9

PROCESSING COMPLETED FOR L9
L10 361 DUP REM L9 (203 DUPLICATES REMOVED)

=> s l10 and gastritis

L11 184 L10 AND GASTRITIS

=> d bib ab 1-

YOU HAVE REQUESTED DATA FROM 184 ANSWERS - CONTINUE? Y/(N):y

L11 ANSWER 1 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 2001:104980 BIOSIS
DN PREV200100104980
TI Association of CagA-positive infection with Helicobacter ***pylori*** ***antibodies*** of IgA class.
AU Rautelin, Hilpi I. K. (1); Oksanen, Aino M.; Karttunen, Riitta A.; Seppala, Kari M. Y.; Virtamo, Jarmo R. K.; Aromaa, Arpo J.; Kosunen, Timo U.
CS (1) Department of Bacteriology and Immunology, University of Helsinki, 00014, Helsinki: Hilpi.Rautelin@Helsinki.fi Finland
SO Annals of Medicine, (December, 2000) Vol. 32, No. 9, pp. 652-656. print. ISSN: 0785-3890.
DT Article
LA English

SL English
AB cagA gene, the best known virulence factor of Helicobacter ***pylori*** , codes for an immunodominant CagA protein. In this study, CagA ***antibodies*** of the IgG class were measured by immunoblot or enzyme ***immunoassay*** in subjects with positive H. ***pylori*** serology, and the presence of CagA ***antibodies*** was compared with that of H. ***pylori*** ***antibodies*** of IgA and IgG classes. Serum samples were available for a total of 1481 subjects, including gastroscopied patients with biopsy-verified H. ***pylori*** infection, smoking men with a normal or low serum ***pepsinogen*** I level indicating atrophic corpus ***gastritis*** , and subjects who later developed ***gastric*** cancer and their matched controls. CagA ***antibodies*** were significantly more prevalent among individuals with elevated H. ***pylori*** ***antibody*** titres of the IgA class than in those with IgG ***antibodies*** only, with the exception of a small subgroup of individuals who later developed ***gastric*** cancer. CagA-positive H. ***pylori*** strains seem to induce an immune response with a markedly higher frequency of IgA than what is found in inflammation caused by CagA-negative strains. The presence of serum IgA ***antibodies*** to H. ***pylori*** seems to indicate a higher risk for CagA-positive H. ***pylori*** infection and possibly more severe late sequelae of the disease.

L11 ANSWER 2 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 2000:506124 BIOSIS
DN PREV200000506124
TI Detection of anti-CagA ***antibodies*** in H. ***pylori*** -infected children by an anti-CagA EIA method.
AU Lopez-Brea, M. (1); Martinez, M. J.; Sanz, J. C. (1); Domingo, D. (1); Alacron, T. (1)
CS (1) Hosp. De La Princesa, Madrid Spain
SO Abstracts of the Interscience Conference on Antimicrobial Agents and Chemotherapy, (1999) Vol. 39, pp. 237. cd-rom.
Meeting Info.: 39th Interscience Conference on Antimicrobial Agents and Chemotherapy San Francisco, California, USA September 26-29, 1999 American Society for Microbiology

DT Conference
LA English
SL English

L11 ANSWER 3 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 2000:442192 BIOSIS
DN PREV200000442192
TI Evaluation of blood tests to predict normal ***gastric*** mucosa.
AU Oksanen, A.; Sipponen, P.; Miettinen, A.; Sarna, S.; Rautelin, H. (1)
CS (1) Dept. of Bacteriology and Immunology, University of Helsinki, FIN-00014, Helsinki Finland
SO Scandinavian Journal of Gastroenterology, (August, 2000) Vol. 35, No. 8, pp. 791-795. print.
ISSN: 0036-5521.
DT Article
LA English
SL English
AB Background: To determine the accuracy of blood tests in predicting normal

gastric mucosa confirmed by histological examination of ***gastric*** biopsy specimens. Methods: In total, 207 consecutive patients referred for upper endoscopy were included. Two biopsy specimens each from the antrum and corpus were assessed histologically for the presence of Helicobacter ***pylori*** , ***gastritis*** , and atrophy. Serum samples were studied for H. ***pylori*** ***antibodies*** by enzyme ***immunoassay*** (Pyloriset EIA-G and EIA-A) and by a rapid latex agglutination test (Pyloriset Dry); ***pepsinogen*** I was measured by an immunoenzymometric assay (Gastroset PGI), ***gastrin*** by radioimmunoassay, and parietal cell ***antibodies*** by indirect immunofluorescence. Results: In 101 (49%) of 207 patients, the ***gastric*** mucosa on histologic examination was normal. In the 63 patients aged 45 years or less, H. ***pylori*** IgG serology was negative in all 47 patients with normal ***gastric*** mucosa and none had low serum ***pepsinogen*** I levels. Among 144 patients over age 45 years, 72 had negative H. ***pylori*** IgG serology. Combining the serum ***pepsinogen*** I assay with the results of H. ***pylori*** IgG serology, 12 patients with normal serology but low serum ***pepsinogen*** I were found. Thus, 60 patients, 52 of whom showed normal ***gastric*** histology, had normal IgG serology and serum ***pepsinogen*** I. In the remaining eight patients with normal blood tests, the histologic changes were very mild. Conclusions: Although negative H. ***pylori*** IgG serology alone in younger patients, and in combination with normal serum ***pepsinogen*** I levels in older patients, reliably predicted the presence of normal ***gastric*** mucosa, gastroscopy is still recommended for patients over 45 years.

L11 ANSWER 4 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 2000:182123 BIOSIS
DN PREV200000182123
TI Atrophic ***gastritis*** and Helicobacter ***pylori*** infection in outpatients referred for gastroscopy.
AU Oksanen, A.; Sipponen, P.; Karttunen, R.; Miettinen, A.; Veijola, L.; Sarna, S.; Rautelin, H. (1)
CS (1) Department of Bacteriology and Immunology, University of Helsinki, FIN-00014, Helsinki Finland
SO Gut, (April, 2000) Vol. 46, No. 4, pp. 460-463.
ISSN: 0017-5749.
DT Article
LA English
SL English
AB Background: Atrophic ***gastritis*** has been shown to be one of the long term sequelae of Helicobacter ***pylori*** infection. Aims: To determine the prevalence of atrophic ***gastritis*** in outpatients, to study the accuracy of serological methods for revealing atrophy, and to define the association of H ***pylori*** infection with atrophic ***gastritis*** in these patients. Patients/methods: A total of 207 consecutive outpatients referred for gastroscopy were included. Biopsy specimens from the antrum and corpus were assessed histologically according to the Sydney system. Serum samples were studied for H ***pylori*** IgG and IgA ***antibodies*** by enzyme ***immunoassay*** , CagA ***antibodies*** by immunoblot, ***pepsinogen*** I by an immunoenzymometric assay, ***gastrin*** by radioimmunoassay, and parietal cell ***antibodies*** by indirect

immunofluorescence. Results: Histological examination revealed atrophic ***gastritis*** in 52 (25%) of 207 patients. H ***pylori*** and CagA ***antibodies*** were strongly associated with atrophic antral ***gastritis*** but poorly associated with atrophic corpus ***gastritis*** . Low serum ***pepsinogen*** I was the most sensitive and specific indicator of moderate and severe atrophic corpus ***gastritis*** . All six patients with moderate atrophic corpus ***gastritis*** had H ***pylori*** infection but eight of 10 patients with severe atrophic corpus had increased parietal cell ***antibodies*** and nine had no signs of H ***pylori*** infection. Conclusions: Atrophic antral ***gastritis*** was strongly associated with CagA positive H ***pylori*** infection. Severe atrophic corpus ***gastritis*** was not determined by H ***pylori*** tests but low serum ***pepsinogen*** I, high ***gastrin*** , and parietal cell ***antibodies*** may be valuable in detecting these changes.

L11 ANSWER 5 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 2000:95018 BIOSIS
DN PREV200000095018
TI Evaluation of rapid ***antibody*** tests for the diagnosis of Helicobacter ***pylori*** infection.
AU Faigel, Douglas O. (1); Magaret, Nathan; Corless, Christopher; Lieberman, David A.; Fennerty, M. Brian
CS (1) Portland VA Medical Center (P3GI), 3710 US Veterans Hospital Road, Portland, OR, 97201 USA
SO American Journal of Gastroenterology, (Jan., 2000) Vol. 95, No. 1, pp. 72-77.
ISSN: 0002-9270.
DT Article
LA English
SL English
AB OBJECTIVE: The aim of this study was to compare the performance characteristics of one serum and four whole blood rapid ***antibody*** tests for Helicobacter ***pylori*** infection. METHODS: A total of 97 outpatients referred for endoscopic evaluation of dyspepsia were included. Antral biopsies were obtained for histology and rapid urease test. Serum was tested with an enzyme-linked ***immunoassay*** (HM-CAP) and a rapid serology test (FlexSure HP). A commercially available 13C-urea breath test was performed. Capillary blood obtained by fingerstick was tested with FlexSure HP, QuickVue, Accustat, and StatSimple ***pylori*** tests. Sensitivity, specificity, and accuracy of each rapid test was calculated relative to a criterion standard of histological ***gastritis*** and at least two of the four following tests positive: identifiable organisms on specially stained slides, rapid urease test, urea breath test, or serum ***immunoassay*** . RESULTS: A total of 30 patients (31%) were infected. The FlexSure HP Serum, and FlexSure HP, QuickVue, Accustat, and StatSimple ***pylori*** whole blood tests had sensitivities of 90%, 87%, 83%, 76%, and 90%; specificities of 94%, 90%, 96%, 96%, and 98%, and accuracies of 93%, 88%, 92%, 87%, and 96%, respectively. Sensitivities were not statistically different. StatSimple ***pylori*** was more specific than FlexSure HP whole blood ($p < 0.03$), and more accurate than FlexSure whole blood ($p < 0.024$) and Accustat ($p < 0.01$). Serum ***immunoassay*** was significantly more sensitive (97%) than FlexSure whole blood, QuickVue, and Accustat ($p < 0.01$), but its specificity (95%) was not statistically different from the rapid tests.

CONCLUSION: Rapid ***antibody*** testing provides an accurate diagnosis of H. ***pylori*** infection. In general, these tests are less sensitive than, but as specific as, standard serology.

L11 ANSWER 6 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1999:309139 BIOSIS
DN PREV199900309139
TI Correlation of serum immunoglobulin G H. ***pylori*** ***antibody*** titers with histologic & endoscopic findings in patients with dyspepsia.
AU Park, E. M. (1); Shim, S. C. (1); Park, C. Y. (1); Shon, J. I. (1); Jun, W. K. (1); Kim, B. I. (1); Jung, E. S. (1); Lee, S. J. (1); Shin, J. H. (1); Keum, J. S. (1)
CS (1) Sungkyunkwan Univ Coll of Medicine, Seoul South Korea
SO Gastroenterology, (April, 1999) Vol. 116, No. 4 PART 2, pp. A277. Meeting Info.: Digestive Disease Week and the 100th Annual Meeting of the American Gastroenterological Association Orlando, Florida, USA May 16-19, 1999 American Gastroenterological Association . ISSN: 0016-5085.
DT Conference
LA English

L11 ANSWER 7 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1999:291593 BIOSIS
DN PREV199900291593
TI Spontaneous decline of H. ***pylori*** ***antibody*** titres in patients with advanced atrophic ***gastritis*** .
AU Kokkola, Arto (1); Puolakkainen, Pauli (1); Rautelin, Hilpi; Sipponen, Pentti; Haapiainen, Reijo (1); Kivilaakso, Eero (1); Kosunen, Timo
CS (1) Dept of Surg, Helsinki Univ Ctral Hosp, Helsinki Finland
SO Gastroenterology, (April, 1999) Vol. 116, No. 4 PART 2, pp. A218. Meeting Info.: Digestive Disease Week and the 100th Annual Meeting of the American Gastroenterological Association Orlando, Florida, USA May 16-19, 1999 American Gastroenterological Association . ISSN: 0016-5085.
DT Conference
LA English

L11 ANSWER 8 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1999:205896 BIOSIS
DN PREV199900205896
TI ***Antibody*** against Helicobacter ***pylori*** CagA and VacA and the risk for ***gastric*** cancer.
AU Yamaoka, Y. (1); Kodama, T.; Kashima, K.; Graham, D. Y.
CS (1) Department of Medicine, Veterans Affairs Medical Center (111D), 2002 Holcombe Blvd, Houston, TX, 77030 USA
SO Journal of Clinical Pathology (London), (March, 1999) Vol. 52, No. 3, pp. 215-218.
ISSN: 0021-9746.
DT Article
LA English
SL English
AB Aim-Helicobacter ***pylori*** is associated with ***gastric*** cancer. Our aim was to investigate whether CagA or VacA seropositivity provides additional risk for ***gastric*** cancer. Methods-Sera from 110 ***gastric*** cancer patients were sex and aged matched with

asymptomatic controls. H ***pylori*** status was determined by IgG enzyme ***immunoassay*** (HM-CAP EIA); CagA status was assessed by enzyme linked immunosorbent assay (ELISA) (OraVax) and immunoblotting (Chiron), and VacA status by immunoblotting using recombinant proteins as antigens. Results-H ***pylori*** infection was associated with an increased risk of ***gastric*** cancer (odds ratio (OR) = 2.19, 95% confidence interval 1.17 to 4.1). Subgroup analysis showed a significant association with intestinal type (OR = 2.94, 1.35 to 6.41), distal type (OR = 2.97, 1.39 to 6.33), early ***gastric*** cancer (OR = 3.74, 1.54 to 9.06), and age ltoreq 55 years (OR = 8.33, 2.04 to 34.08), but not with diffuse type (OR = 0.83), proximal type (OR = 1.0), advanced ***gastric*** cancer (OR = 1.13), or age > 55 years (OR = 1.40). Serum CagA IgG and VacA ***antibody*** positivity was present in similar proportions in patients with and without cancer, with no significant differences in histological classification, clinical stage, or location (p > 0.3). Conclusions-H ***pylori*** infection causes chronic ***gastritis*** and is associated with the development of ***gastric*** cancer. Neither CagA nor VacA seropositivity added additional information or stratification.

L11 ANSWER 9 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1999:156989 BIOSIS
DN PREV199900156989
TI Detection of Helicobacter ***pylori*** in ***gastric*** biopsies by PCR: Correlation with conventional methods.
AU Polidorou, F. (1); Vavatsi, N.; Pavlitou, K. (1); Malaka, E. (1)
CS (1) Dep. Microbiol., Gen. Hosp. "Agios Dimitrios", Thessaloniki Greece
SO Deltion Ellenikes Mikrobiologikes Etaireias, (May-June, 1998) Vol. 43, No. 3, pp. 276-279.
ISSN: 0438-9573.
DT Article
LA Greek
SL Greek; English
AB The aim of the study was to evaluate the role of PCR technique in the detection of Helicobacter ***pylori*** (HP) in ***gastric*** biopsies. Antral biopsies from 27 patients with chronic ***gastritis*** and peptic ulcer were examined by means of a rapid urease test, Giemsa and haematoxylin-eosin stains and PCR. Blood samples were tested for IgG ***antibodies*** against HP using an enzyme ***immunoassay*** . After the extraction of DNA, nested PCR was performed by using 2 pairs of primers for the amplification of the urease A gene of HP. The final PCR product was 361bp and it was recognised by electrophoresis on a 2% agarose gel. Nineteen out of 27 patients were positive for HP using PCR. The staining method and the ***immunoassay*** detected HP in 18 patients while CLOtest was positive in 17 patients. In one patient HP was detected only by PCR.

L11 ANSWER 10 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1999:60264 BIOSIS
DN PREV199900060264
TI Severe ***gastritis*** in guinea-pigs infected with Helicobacter ***pylori*** .
AU Sturegard, E.; Sjunnesson, H.; Ho, B.; Willen, R.; Aleljung, P.; Ng, H. C.; Wadstrom, T. (1)
CS (1) Dep. Med. Microbiol., Lund Univ., Lund Sweden

SO Journal of Medical Microbiology, (Dec., 1998) Vol. 47, No. 12, pp. 1123-1129.
ISSN: 0022-2615.
DT Article
LA English
AB An appropriate animal model is essential to study Helicobacter ***pylori*** infection. The aim of this study was to investigate if H. ***pylori*** can colonise the guinea-pig stomach and whether the infection causes ***gastritis*** and a serological response similar to that observed in man. Guinea-pigs were infected either with fresh H. ***pylori*** isolates from human ***gastric*** biopsies or with a guinea-pig passaged strain. When the animals were killed, 3 and 7 weeks after inoculation, samples were taken for culture, histopathology and serology. H. ***pylori*** was cultured from 22 of 29 challenged animals. All culture-positive animals exhibited a specific immune response against H. ***pylori*** antigens in Western blotting and ***gastritis*** in histopathological examination. ***Antibody*** titres in enzyme ***immunoassay*** were elevated among animals challenged with H. ***pylori*** . The inflammatory response was graded as severe in most animals and consisted of both polymorphonuclear leucocytes and lymphocytes. Erosion of the ***gastric*** epithelium was found in infected animals. These results suggest that the guinea-pig is suitable for studying H. ***pylori*** -associated diseases. Moreover, guinea-pigs are probably more similar to man than any other small laboratory animal as regards ***gastric*** anatomy and physiology.

L11 ANSWER 11 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1999:39237 BIOSIS
DN PREV199900039237
TI Serodiagnosis of Helicobacter ***pylori*** infection in Korean patients using enzyme-linked immunosorbent assay.
AU Kim, S. Y.; Ahn, J. S.; Ha, Y. J.; Doh, H. J.; Jang, M. H.; Chung, S. L.; Park, H. J. (1)
CS (1) MOGAM Biotechnology Res. Inst., 341 Pojung-Ri, Koosung-Myon, Yongin-City, Kyonggi-Do 449-910 South Korea
SO Journal of Immunoassay, (Nov., 1998) Vol. 19, No. 4, pp. 251-270.
ISSN: 0197-1522.
DT Article
LA English
AB Helicobacter ***pylori*** (H. ***pylori***) is a gram-negative spiral bacteria that are associated with ***gastritis*** , peptic ulcer and ***gastric*** cancer. We have developed enzyme-linked immunosorbent assay (ELISA) that detects serum anti-H. ***pylori*** immunoglobulin G ***antibodies*** using H. ***pylori*** strains isolated from Korean patients. To assess the sensitivity and specificity of our assay system with different commercial kits, serum samples from 249 Korean patients with a variety of gastrointestinal diseases were tested. Among 249 Korean patients, 178 (71.5%) were positive in culture and/or urease test. The sensitivity and specificity between our assay system and four other commercial kits (Bio-Rad, DAKO, ROCHE, and IPR) were as follows: 97.8% and 92%, 94.3% and 53%, 56.5% and 92%, 83.3% and 96%, 58.2% and 92%, respectively. All sera showing discordant ***immunoassay*** results between different ELISA kits were confirmed by immunoblot analysis. These results indicate that our assay system showed a highly

accurate and reliable results in diagnosis of H. ***pylori*** infection in Korean patients.

L11 ANSWER 12 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1998:445623 BIOSIS
DN PREV199800445623
TI Helicobacter ***pylori*** -positive ***gastritis*** in pediatric patients with chronic inflammatory bowel disease.
AU Kolho, Kaija-Leera; Rautelin, Hilpi; Lindahl, Harry; Savilahti, Erkki (1)
CS (1) Hosp. Children Adolescents, Stenbackinkatu 11, FIN-00290 Helsinki Finland
SO JPGN, (Sept., 1998) Vol. 27, No. 3, pp. 292-295.
DT Article
LA English
AB Background: ***Gastritis*** is a common finding in patients with inflammatory bowel disease. However, the association of ***gastritis*** with Helicobacter ***pylori*** is unclear in these patients. Methods: The prevalence of ***antibodies*** for H. ***pylori*** in serum was determined in 47 pediatric patients with inflammatory bowel disease (19 with Crohn's disease, 21 with ulcerative colitis, and 7 with unclassified disease). H. ***pylori*** ***antibodies*** of the IgG and IgA classes were measured by enzyme ***immunoassay*** in 24 patients at the time of diagnosis of inflammatory bowel disease and in 23 more patients during the follow-up of inflammatory bowel disease (mean follow-up, 3.5 years; range 1-10 years). Esophagogastroduodenoscopy was performed on 40 patients during the examination for inflammatory bowel disease. Results: In contrast to earlier findings, no patient was determined to be positive for H. ***pylori*** , either in serologic or histologic examination. This negative finding was unexpected, because it is known that approximately 10% of asymptomatic Finnish children have ***antibodies*** for H. ***pylori*** in serum and approximately 10% of analyses of specimens obtained in ***gastric*** antral biopsies obtained at the Hospital for Children and Adolescents, Helsinki, Finland, are positive for H. ***pylori*** . Conclusions: Permanent colonization of the stomach with H. ***pylori*** is unusual in children with inflammatory bowel disease.

L11 ANSWER 13 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1998:304689 BIOSIS
DN PREV199800304689
TI Positive result by serology indicates active Helicobacter ***pylori*** infection in patients with atrophic ***gastritis*** .
AU Kokkola, Arto (1); Rautelin, Hilpi; Puolakkainen, Pauli; Sipponen, Pentti; Farkkila, Martti; Haapiainen, Reijo; Kosunen, Timo U.
CS (1) Second Dep. Surgery, Helsinki Univ. Central Hosp., Haartmaninkatu 4, FI-00290 Helsinki Finland
SO Journal of Clinical Microbiology, (June, 1998) Vol. 36, No. 6, pp. 1808-1810.
ISSN: 0095-1137.
DT Article
LA English
AB Patients with atrophic corpus ***gastritis*** and elevated Helicobacter ***pylori*** ***antibody*** titers but 130 -urea breath test (13C-UBT) and histology results negative for H. ***pylori*** were randomized into eradication therapy or follow-up only.

Antibody levels decreased significantly in six out of seven patients in the eradication group, while in the follow-up group, the titers declined in only one out of eight patients. In patients with atrophic corpus ***gastritis*** , positive serology results may indicate an ongoing infection in spite of negative 13C-UBT and histology results.

L11 ANSWER 14 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1998:229535 BIOSIS
DN PREV199800229535
TI Evaluation of pyloriset screen, a rapid whole-blood diagnostic test for Helicobacter ***pylori*** infection.
AU Oksanen, Aino; Veijola, Lea; Sipponen, Pentti; Schauman, Knut-Olof; Rautelin, Hilpi (1)
CS (1) Dep. Bacteriol. Immunol., P.O. Box 21, Univ. Helsinki, Helsinki FIN-00014 Finland
SO Journal of Clinical Microbiology, (April, 1998) Vol. 36, No. 4, pp. 955-957.
ISSN: 0095-1137.
DT Article
LA English
AB Helicobacter ***pylori*** infection can be detected by several invasive tests based on gastroscopy and by noninvasive methods such as serologic assays. Noninvasive tests can be used not only in addition to invasive tests but also by themselves to screen for H. ***pylori*** infection in patients who are not in urgent need of endoscopy. Lately, rapid qualitative serologic tests have been developed. In the present study, the accuracy of a novel rapid whole-blood test, Pyloriset Screen, detecting immunoglobulin G (IgG) and IgA ***antibodies*** against H. ***pylori*** was evaluated. A total of 207 consecutive adult outpatients referred for upper endoscopy were enrolled. ***Gastric*** biopsy specimens were taken from the antrum and corpus for histologic examination and rapid urease testing. Cultures were available for 113 patients. Serum samples collected from all patients were tested for H. ***pylori*** ***antibodies*** by two enzyme ***immunoassays*** (EIAs) (Pyloriset EIA and an in-house EIA), a rapid latex agglutination test (Pyloriset Dry), and Pyloriset Screen. Patients were considered H. ***pylori*** positive if helicobacters were seen on histologic examination (77 patients) or, if in combination with histologically verified (although helicobacter-negative) ***gastritis*** , their IgG ***antibody*** titers were elevated in the two EIAs (five patients). The Pyloriset Screen test had a sensitivity of 95%, a specificity of 94%, a positive predictive value of 91%, and a negative predictive value of 97%. Among 63 patients under the age of 45 years, the Pyloriset Screen test did not miss a single H. ***pylori*** diagnosis, and only 1 patient had a false-positive result. Pyloriset Screen could be used reliably to screen for H. ***pylori*** infection.

L11 ANSWER 15 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1998:167615 BIOSIS
DN PREV199800167615
TI A new histological procedure for re-evaluation of the serologial test for Helicobacter ***pylori*** .
AU Misawa, K. (1); Kumagai, T.; Shimizu, T.; Furihata, K.; Ota, H.; Akamatsu, T.; Katsuyama, T.

CS (1) Dep. Lab. Med., Shinshu Univ. Med., Asahi 3-1-1 Matsumoto 390 Japan
SO European Journal of Clinical Microbiology & Infectious Diseases, (Jan., 1998) Vol. 17, No. 1, pp. 14-19.
ISSN: 0934-9723.
DT Article
LA English
AB To re-evaluate the accuracy of the serological test for Helicobacter ***pylori*** , fixation of biopsy specimens with Carnoy's solution (preserving the mucous layer in tissue preparations) followed by immunohistochemical staining (a new histological procedure) was used as the reference histological method instead of 10% formalin fixation followed by hematoxylin-eosin staining (the conventional histological procedure). Biopsy specimens (antrum and body) from 114 patients with ***gastritis*** (including non-ulcer dyspepsia) or ***gastric*** and/or duodenal ulcers were obtained by endoscopy and used for both bacteriological culture and histological examination. Serum samples were taken from all patients at the time of endoscopy. The serum levels of specific IgG and IgA ***antibodies*** for Helicobacter ***pylori*** were measured by commercial enzyme ***immunoassay*** kits. The reliability of the IgG and IgA measurements was evaluated by analyzing receiver operating characteristic curves obtained using the two histological procedures. With the conventional histological procedure as the reference, the sensitivity and specificity levels of the serological test were 87.2% and 82.1%, respectively. With the new histological procedure as reference, sensitivity and specificity were 94% and 96.7%, respectively. The insufficient accuracy reported for the serological test could be due to false-positive or false-negative results obtained when the conventional histological procedure is used as the reference. The new histological procedure used here revealed that the serological test for Helicobacter ***pylori*** is more reliable than previously thought.

L11 ANSWER 16 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1997:501874 BIOSIS
DN PREV199799801077
TI Reactivities of Lewis antigen monoclonal ***antibodies*** with the lipopolysaccharides of Helicobacter ***pylori*** strains isolated from patients with gastroduodenal diseases in Japan.
AU Amano, Ken-Ichi (1); Hayashi, Shyunji; Kubota, Toru; Fujii, Nobuhiro; Yokota, Shin-Ichi
CS (1) Central Res. Lab., Akita Univ. Sch. Med., 1-1-1 Hondo, Akita, Akita 010 Japan
SO Clinical and Diagnostic Laboratory Immunology, (1997) Vol. 4, No. 5, pp. 540-544.
ISSN: 1071-412X.
DT Article
LA English
AB We have examined the reactivity of monoclonal ***antibodies*** (Mabs) specific for Lewis antigens (Le-x, Le-y, Le-a, and Le-b) with Helicobacter ***pylori*** lipopolysaccharides (LPS) by immunoblot analysis and enzyme-linked immunosorbent assay (ELISA). Sixty-eight strains of H. ***pylori*** were isolated from patients with chronic ***gastritis*** , ***gastric*** and duodenal ulcers, and ***gastric*** cancer in Japan. The cells were treated with proteinase K, and the resulting fractions were used as a source of LPS for the ***immunoassays*** . In the immunoblot analysis, 28 isolates (41%) and 29 isolates (42%) reacted

with anti-Le-x and anti-Le-y MAbs, respectively, while 4 isolates (6%) and 7 isolates (10%) reacted with anti-Le-b and anti-Le-b MAbs. On the other hand, in ELISA, the number of isolates that reacted with anti-Le-x MAbs fell significantly to 21 isolates (30%) but the number of isolates that reacted with the other anti-Lewis antigen MAbs remained relatively unchanged. These data show that the immunoblotting technique is more sensitive than the ELISA technique for the detection of immunocomplexes of anti-Le-x MAbs and components of H. ***pylori*** LPS. Furthermore, human serum was found to react with the synthetic Lewis antigens regardless of the status of the individual's H. ***pylori*** infection. This means that humans may naturally possess ***antibodies*** against Lewis antigens in the absence of H. ***pylori*** infection.

L11 ANSWER 17 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1997:35048 BIOSIS
DN PREV199799341451
TI Seroprevalence of Helicobacter ***pylori*** in south Sweden and Iceland.
AU Bergenzaun, P.; Kristinsson, K. G.; Thjodleifsson, B.; Sigvaldadottir, E.; Molstad, S.; Held, M.; Wadstrom, T. (1)
CS (1) Dep. Medical Microbiol., Lund University, Solvegatan 23, S-22362 Lund Sweden
SO Scandinavian Journal of Gastroenterology, (1996) Vol. 31, No. 12, pp. 1157-1161.
ISSN: 0036-5521.
DT Article
LA English
AB Background: Seroepidemiologic studies on the prevalence of Helicobacter ***pylori*** infection have been reported from several European countries but not from Sweden or Iceland. Methods: Serum samples were collected from 443 persons in Sweden and 387 persons in Iceland. All the 830 sera were tested with the same enzyme ***immunoassay*** test with an acid glycine extract of H. ***pylori*** surface proteins as antigen. Results: The ***antibody*** levels were low in the young age groups in both Sweden and Iceland, with increasing levels with age. Conclusions: The results are consistent with previous studies from other comparable countries, but with important differences. The prevalence was lower in Sweden than in other, previously studied, Western European countries, but, on the other hand, the prevalence was slightly higher in Iceland.

L11 ANSWER 18 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1996:269657 BIOSIS
DN PREV199698825786
TI Evaluation of three commercial enzyme ***immunoassays*** compared with the 13C urea breath test for detection of Helicobacter ***pylori*** infection.
AU Marchildon, P. A. (1); Ciota, L. M.; Zamaniyan, F. Z.; Peacock, J. S.; Graham, D. Y.
CS (1) Enteric Products Inc., 25 E. Loop Rd., Stony Brook, NY 11790 USA
SO Journal of Clinical Microbiology, (1996) Vol. 34, No. 5, pp. 1147-1152.
ISSN: 0095-1137.
DT Article
LA English
AB The diagnostic significance of the serological detection of

antibodies to Helicobacter ***pylori*** has been established by numerous investigators. Reports of the clinical reliabilities of commercial enzyme ***immunoassay*** (EIA) kits available for this purpose vary as a result of the different H. ***pylori*** antigen sources and reference methods used. The 13C urea breath test (UBT) has been shown to be an extremely accurate and reliable method of detecting H. ***pylori*** infection. We used the 13C urea breath test as the confirmatory method for H. ***pylori*** status to evaluate three commercially available EIA kits designed to detect immunoglobulin G ***antibodies*** to H. ***pylori*** . These kits were the HM-CAP EIA kit (Enteric Products, Inc.), the ***PYLORI*** STAT EIA kit (BioWhittaker, Inc.), and the G.A.P. kit (Bio-Rad Laboratories/Biomerica, Inc.). The evaluations were performed in a double-blind manner with samples from 473 clinically characterized patients. This group included patients with symptomatic gastrointestinal disorders as well as nonsymptomatic volunteers. The sensitivities of the kits were as follows: HM-CAP, 98.4%; ***PYLORI*** STAT, 99.2%; and GA.P., 100%. The specificities were as follows: HM-CAP, 96.4%; ***PYLORI*** STAT, 90.1%; and G.A.P., 26.0%. Although the HM-CAP and ***PYLORI*** STAT kits performed comparably, the G.A.P. test yielded significantly more false-positive results and an unacceptably high number of indeterminate results.

L11 ANSWER 19 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1996:221204 BIOSIS
DN PREV199698777333
TI High frequency of Helicobacter negative ***gastritis*** in patients with Crohn's disease.
AU Halme, L. (1); Karkkainen, P.; Rautelin, H.; Kosunen, T. U.; Sipponen, P.
CS (1) Dep. Surg., Helsinki Univ. Hosp., Kasarmikatu 11-13, FIN-00130 Helsinki Finland
SO Gut, (1996) Vol. 38, No. 3, pp. 379-383.
ISSN: 0017-5749.
DT Article
LA English
AB The frequency of ***gastric*** Crohn's disease has been considered low. This study was undertaken to determine the prevalence of chronic ***gastritis*** and Helicobacter ***pylori*** infection in patients with Crohn's disease. Oesophagogastroduodenoscopy was performed on 62 consecutive patients suffering from ileocolonic Crohn's disease. Biopsy specimens from the antrum and corpus were processed for both histological and bacteriological examinations. H. ***pylori*** ***antibodies*** of IgG and IgA classes were measured in serum samples by enzyme ***immunoassay*** . Six patients (9.7%) were infected with H. ***pylori*** , as shown by histology, and in five of them the infection was also verified by serology. Twenty one patients (32%) had chronic H. ***pylori*** negative ***gastritis*** (negative by both histology and serology) and one of them also had atrophy in the antrum and corpus. Granulomas were found in four patients. The characteristic appearance of H. ***pylori*** negative ***gastritis*** was focal and mostly mild inflammation resembling the inflammatory changes seen in the gut in Crohn's disease. Patients with H. ***pylori*** negative chronic ***gastritis*** had a significantly more active disease in their gut than those with normal ***gastric*** mucosa (p lt 0.01). It is concluded that H. ***pylori*** positive ***gastritis*** is rare,

while H. ***pylori*** negative ***gastritis*** is relatively common in patients with Crohn's disease. H. ***pylori*** negative 'Crohn's ***gastritis*** ' seems to be associated with active Crohn's disease.

L11 ANSWER 20 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1995:550325 BIOSIS
DN PREV199698564625
TI ***Antibody*** titres in Helicobacter ***pylori*** infection: Implications in the follow-up of antimicrobial therapy.
AU Kosunen, Timo U.
CS Dep. Bacteriology Immunology, P.O. Box 21, FIN-00014 Univ. Helsinki, Helsinki Finland
SO Annals of Medicine, (1995) Vol. 27, No. 5, pp. 605-607. ISSN: 0785-3890.
DT Article
LA English
AB Regular presence and persistence of specific serum ***antibodies*** in Helicobacter ***pylori*** infection gives an excellent tool for diagnostic work. Eradication of the infection leads to gradual disappearance of the ***gastritis*** and decrease of specific serum ***antibodies*** . The fall of IgG and IgA ***antibody*** titres can be followed with quantititative enzyme ***immunoassays*** . Success in eradication is reflected in 40-50% decrease of ***antibody*** titres within 5-6 months. The decrease continues and most patients have normal titres within 2 years. Serology offers a cheap and convenient way to follow the treated patients and makes most follow-up endoscopies unnecessary.

L11 ANSWER 21 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1995:439431 BIOSIS
DN PREV199598453731
TI Helicobacter ***pylori*** infection in recurrent abdominal pain in childhood: Comparison of diagnostic tests and therapy.
AU Chong, Sonny K. F. (1); Lou, Qinyuan; Asnicar, Mark A.; Zimmerman, Sarah E.; Croffie, Joseph M.; Lee, Chao-Hung; Fitzgerald, Joseph F.
CS (1) Dep. of Pediatrics, James Whitcomb Riley Hospital for Children, Indiana Univ. School of Med., 702 Barnhill Dr., Room 2728, Indianapolis, IN 46202-5225 USA
SO Pediatrics, (1995) Vol. 96, No. 2 PART 1, pp. 211-215. ISSN: 0031-4005.
DT Article
LA English
AB Objective: To determine the role of Helicobacter ***pylori*** infection in children with recurrent abdominal pain and the usefulness of serologic tests in screening H. ***pylori*** infection and monitoring treatment of H. ***pylori*** -associated ***gastritis*** . Methods: During a 3 year period, we investigated the presence of serum immunoglobulin G (IgG) ***antibody*** to H. ***pylori*** in 456 children using the high-molecular-weight cell-associated protein H. ***pylori*** enzyme ***immunoassay*** kit. Among the 456 children studied, 218 (age range, 3 to 18 years; mean age, 9.5 years) had symptoms of recurrent abdominal pain (RAP syndrome) with or without vomiting, and the remaining 238 (age range, 3 to 18 years; mean age, 9.8 years) had no RAP (non-RAP syndrome). We performed upper gastrointestinal endoscopy on

111 consecutive children of the 218 with RAP syndrome and obtained mucosal biopsies for culture, histologic analysis, CLO test (Delta West, Perth, Australia), and H. ***pylori*** detection by polymerase chain reaction. Results: Thirty-eight (17.4%) of 218 children in the RAP group and 25 (10.5%) of 238 children in the non-RAP group were seropositive for H. ***pylori*** . Of the 111 children endoscoped, 95 were found to be negative, and 12 were positive by all five assays. Specimens from 2 children were negative by culture and the CLO test but positive by the other three assays. Specimens from 1 child were negative by histologic analysis but positive by all other tests. The remaining child was positive for anti-H. ***pylori*** IgG but negative by all of the other four assays. Upper gastrointestinal endoscopy detected 14 children with peptic ulcer disease (9 duodenal ulcer and 5 ***gastric*** ulcer) and 12 with antral nodular ***gastritis*** . Only 4 of the 14 diagnosed with peptic ulcer were H. ***pylori*** positive by all five assays, whereas all 12 children with antral nodular ***gastritis*** were H. ***pylori*** positive. Nine of the 12 H. ***pylori*** -positive children were treated with a combination of bismuth subsalicylate, amoxicillin, and metronidazole for 2 weeks. Sera obtained at 2, 4, and 6 months after treatment from all 9 children showed a decrease in anti-H. ***pylori*** IgG titer. Three H. ***pylori*** -infected children who did not receive any treatment served as control children, and their IgG levels remained elevated or increased over time. Conclusion: The results from our study indicate that screening for the serum IgG ***antibody*** to H. ***pylori*** is a practical method for diagnosing H. ***pylori*** infection in children, and that serial measurements of the H. ***pylori*** IgG ***antibody*** are useful for monitoring treatment of H. ***pylori*** because of its high sensitivity and ease of performance. Only 4 of the 14 children diagnosed with peptic ulcer disease were confirmed to be infected with H. ***pylori*** , whereas all 12 children with antral nodular ***gastritis*** were found to be infected by H. ***pylori*** . These observations suggest that H. ***pylori*** infection is more frequently associated with ***gastritis*** than with peptic ulcer disease in children, and that H. ***pylori*** ***gastritis*** is a cause of RAP syndrome in children.

L11 ANSWER 22 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1995:245551 BIOSIS
DN PREV199598259851
TI Evaluation of an Enzyme ***Immunoassay*** (Helisal) for Detection of Salivary ***Antibody*** to Helicobacter ***pylori*** .
AU Lin, E. (1); Simor, A. E.; Pearen, S.; Saibil, F.; Cohen, L.; Hung, S.; Donhoffer, H. A.; Louie, M.
CS (1) Sunnybrook Health Sci. Cent., Univ. Toronto, Toronto, ON Canada
SO Gastroenterology, (1995) Vol. 108, No. 4 SUPPL., pp. A150.
Meeting Info.: 95th Annual Meeting of the American Gastroenterological Association and Digestive Disease Week San Diego, California, USA May 14-17, 1995
ISSN: 0016-5085.
DT Conference
LA English

L11 ANSWER 23 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1995:228024 BIOSIS
DN PREV199598242324

TI Helicobacter ***pylori*** infection in rural settlements (Kibbutzim) in Israel.
AU Gilboa, S. (1); Gabay, G.; Zamir, D.; Zeev, A.; Novis, B.
CS (1) Epidemiol. Unit, Meir Hosp., Kfar-Sava 44281 Israel
SO International Journal of Epidemiology, (1995) Vol. 24, No. 1, pp. 232-237. ISSN: 0300-5771.
DT Article
LA English
AB Background: Helicobacter ***pylori*** (HP) is accepted as a major cause of type B ***gastritis*** , which is strongly associated with peptic ulcer disease. Epidemiological studies have indicated a correlation of HP infection and socioeconomic class. Methods: To determine the prevalence of HP infection and to evaluate symptoms and risk factors associated with HP infection in a rural population, 377 asymptomatic individuals were studied out of a random sample of 453 people. Subjects were randomly chosen in a ratio of 1:4 of all the adults over 30 years, living in eight communal settlements in Israel. Blood samples were taken and subjects answered a questionnaire in which sociodemographic information. clinical gastrointestinal background and the use of medication were included. A sensitive enzyme-linked ***immunoassay*** was used to determine ***antibodies*** to HP in serum. Results: The overall prevalence of HP infection was 72%. In a multivariant discriminant analysis: age, country of origin and ethnic group were found to be the most closely associated variables for HP infection and the discriminant analysis succeeded in predicting correctly, in 62% of the population, whether they had or did not have HP infection. There was no significant difference with gender, occupation, educational level, blood group, smoking, gastrointestinal symptoms and use of medication. Conclusions: The prevalence of HP infection was higher than that in industrialized countries, but lower than in developing countries. The prevalence in a rural population was slightly higher than that of an urban population in Israel (65%). The country of origin and ethnic group influenced the prevalence of HP infection and not birth and growing up on the Kibbutz.

L11 ANSWER 24 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1994:392492 BIOSIS
DN PREV199497405492
TI A new method for detecting anti Helicobacter ***pylori*** ***antibodies*** : An analytical and clinical evaluation.
AU Plebani, M. (1); Basso, D.; Brigato, L.; Cassaro, M.; Farinati, F.; Di Mario, F.; Rugge, M.
CS (1) Ist. Med. Lab., c/o Lab. Centrale, Via Giustiniani 2, 35128 Padova Italy
SO Journal of Clinical Laboratory Analysis, (1994) Vol. 8, No. 4, pp. 219-222.
ISSN: 0887-8013.
DT Article
LA English
AB The diagnosis of Helicobacter ***pylori*** (Hp) infection is an important goal in clinical practice. In this paper we evaluated 1. the analytical reliability of a new second-generation antigen based enzyme ***immunoassay*** (Cobas Core Anti Helicobacter ***pylori*** EIA) in detecting anti-Hp IgG ***antibodies*** , and 2. the behaviour of anti-HP IgG in patients with chronic atrophic and non-atrophic ***gastritis*** as compared to healthy controls. The findings from the

dilution curve, the values of intra and inter assay coefficients of variations (never above 10%) and of the recovery test (between 96 and 109%), confirm that the method is reliable. Serum IgG anti-Hp levels were found to be significantly higher in patients with histologically identified Hp infection, than in those negative at histology. Furthermore, the grade of histological positivity was correlated with serum IgG levels. However, we found a discrepancy between a low prevalence of Hp staining and a high prevalence of Hp seropositivity in patients with chronic atrophic or non-atrophic ***gastritis*** , but not in controls. This suggests that IgG serum determination may be more useful than histology in determining a present or previous infection in patients with chronic atrophic or non-atrophic ***gastritis*** .

L11 ANSWER 25 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1994:359852 BIOSIS
DN PREV199497372852
TI Comparison of PCR and other diagnostic techniques for detection of Helicobacter ***pylori*** infection in dyspeptic patients.
AU Weiss, Judith (1); Mecca, James; Da Silva, Elvira; Gassner, Dieter
CS (1) Dep. Infect. Dis., Roche Mol. Systems, Alameda, CA 94501 USA
SO Journal of Clinical Microbiology, (1994) Vol. 32, No. 7, pp. 1663-1668. ISSN: 0095-1137.
DT Article
LA English
AB A sensitive and specific PCR-based assay to detect the Helicobacter ***pylori*** 16S rRNA gene present in formalin-fixed paraffin-embedded ***gastric*** biopsy specimens has been developed. A total of 95 patients with dyspepsia were evaluated for the presence of chronic active ***gastritis*** and an infection with H. ***pylori*** through the use of diagnostic assays based on biopsy specimens and serology. The "gold standard" for the presence of the bacteria was direct detection in histological sections of biopsy specimens by Giemsa stain. The results obtained with the PCR assay performed on the biopsy specimens (94% sensitivity and 100% specificity) were equivalent to the detection of H. ***pylori*** immunoglobulin G ***antibodies*** by the commercially available second-generation Cobas Core anti-H. ***pylori*** immunoglobulin G enzyme ***immunoassay*** (94% sensitivity and 98% specificity) for the diagnosis of H. ***pylori*** infection. Urease testing and bacterial culture of the biopsy specimens were inferior (88 and 70% sensitivity and 96 and 98% specificity, respectively). A Western blot (immunoblot) analysis had slightly greater sensitivity (96%), although specificity was reduced to 93%. This research prototype PCR assay was shown to be highly reliable for the detection of infection with H. ***pylori*** and the presence of chronic active ***gastritis*** in the population studied.

L11 ANSWER 26 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1994:78004 BIOSIS
DN PREV199497091004
TI Evaluation of a new immunodiagnostic assay for Helicobacter ***pylori*** ***antibody*** detection: Correlation with histopathological and microbiological results.
AU Pronovost, Allan D. (1); Rose, Steven L.; Pawlak, Jan W.; Robin, Howard; Schneider, R.
CS (1) Quidel Corp., 10165 McKellar Court, San Diego, CA 92121 USA

SO Journal of Clinical Microbiology, (1994) Vol. 32, No. 1, pp. 46-50.
ISSN: 0095-1137.
DT Article
LA English
AB Infection with Helicobacter ***pylori*** has been associated with the pathogenesis of chronic active ***gastritis*** and ***gastric*** and duodenal ulcer disease. Detection of immunoglobulin G ***antibodies*** to H. ***pylori*** offers a simple alternative to direct detection of the organism in biopsied tissue by culture or histopathological methods. A rapid flow-through membrane-based enzyme ***immunoassay*** for the detection of human immunoglobulin G ***antibodies*** to H. ***pylori*** has been developed and evaluated. Clinical evaluations were performed with 256 patient serum samples obtained from four clinical sites. Biopsy samples were obtained by endoscopic procedures at the same time as the serum samples, and were histopathologically and microbiologically categorized for the presence or absence of H. ***pylori*** . Sensitivity and specificity for this rapid enzyme ***immunoassay*** were 92 and 88%, respectively, compared directly with endoscopy results. After discordant results were resolved by a quantitative microwell enzyme-linked immunosorbent assay, the resulting sensitivity and specificity were 94 and gt 99%, respectively. These results indicate that this rapid enzyme ***immunoassay*** is a useful technique to determine H. ***pylori*** infection status and is a viable alternative to invasive endoscopic procedures.

L11 ANSWER 27 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1993:423557 BIOSIS
DN PREV199345071182
TI Rapid test for detection of ***antibodies*** to Helicobacter ***pylori*** .
AU Anderson, G.; Alemohammad, M. M.; Foley, T. J.; Colletti, A.; Patel, A.; Dooley, C. P.
CS Hycor Biomed. Inc., 7272 Chapman Ave., Garden Grove, CA 92641 USA
SO Clinical Infectious Diseases, (1993) Vol. 16, No. SUPPL. 4, pp. S416-S417.
Meeting Info.: First North American Congress on Anaerobic Bacteria and Anaerobic Infections Marina del Rey, California, USA July 24-26, 1992
ISSN: 1058-4838.
DT Article
LA English

L11 ANSWER 28 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1993:344308 BIOSIS
DN PREV199396041308
TI Diagnosis of Helicobacter ***pylori*** infection by using Pyloriset EIA-G and EIA-A for detection of serum immunoglobulin G (IgG) and IgA ***antibodies*** .
AU Granberg, Christer (1); Mansikka, Antti; Lehtonen, Olli-Pekka; Kujari, Harry; Gronfors, Reijo; Nurmi, Heimo; Raihae, Ismo (1); Stahlberg, Marja-Riitta; Leino, Rauli
CS (1) Orion Corp., Orion Diagnostica, SF-02101 Espoo Finland
SO Journal of Clinical Microbiology, (1993) Vol. 31, No. 6, pp. 1450-1453.
ISSN: 0095-1137.
DT Article
LA English
AB We evaluated the performance of new enzyme ***immunoassay*** (EIA)

kits (Pyloriset; Orion Corporation, Orion Diagnostica, Espoo, Finland) for the detection of immunoglobulin G (IgG) and IgA ***antibodies*** to Helicobacter ***pylori*** in serum. Serum samples from 195 patients with upper abdominal complaints were collected. Biopsy specimens of the ***gastric*** mucosae were taken for histological analysis and bacterial culture. The sensitivity, specificity, positive and negative predictive values, and efficacy of the Pyloriset EIA-G in detecting IgG ***antibodies*** to H. ***pylori*** were 92, 84, 88, 90, and 89%, respectively, when compared with those of the reference methods used. The corresponding data for detection of IgA ***antibodies*** were 80, 89, 89, 79, and 84%, respectively. The overall prevalence of defined H. ***pylori*** positivity was 54%. Moreover, the ***antibody*** tests showed a very good correlation with the biopsy findings. IgG ***antibodies*** were found in 93% of sera from patients with documented ***gastritis*** and H. ***pylori*** positivity, whereas only 4% of the sera from patients with documented ***gastritis*** and H. ***pylori*** -negative patients was positive. The results obtained for IgA ***antibodies*** were 81 and 6%, respectively. We conclude that the Pyloriset EIA-G, the test for IgG ***antibodies*** , is a good and reliable test for the detection of ***antibodies*** to H. ***pylori*** and as an indication of H. ***pylori*** infection. The determination of IgA ***antibodies*** may be used as a test that complements the IgG ***antibody*** assay.

L11 ANSWER 29 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1992:524993 BIOSIS
DN BA94:133068
TI SERODIAGNOSIS OF HELICOBACTER- ***PYLORI*** INFECTIONS WITH AN ENZYME ***IMMUNOASSAY*** USING THE CHROMATOGRAPHICALLY PURIFIED 120 KILODALTON PROTEIN.
AU GERSTENECKER B; ESCHWEILER B; VOEGELE H; KOCH H K; HELLERICH U; KIST M
CS INST. MED. MICROBIOL., UNIV. FREIBURG, HERMANN-HERDER-STR. 11, 7800 FREIBURG, GER.
SO EUR J CLIN MICROBIOL INFECT DIS, (1992) 11 (7), 595-601.
CODEN: EJCDEU. ISSN: 0934-9723.
FS BA; OLD
LA English
AB A membrane-associated 120 kDa protein on Helicobacter ***pylori*** with known species-specificity was isolated and used in an enzyme ***immunoassay*** (EIA) for the detection of Helicobacter ***pylori*** -specific IgG ***antibodies*** in patient sera. The EIA was compared with two other methods used for serodiagnosis of Helicobacter ***pylori*** infections: an EIA using sonicated whole Helicobacter ***pylori*** cell antigen and Western immunoblot. In a pospective study 127 unselected patients (76 patients with antrum ***gastritis*** , 51 patients without ***gastritis***) who underwent gastroscopy were studied histologically and serologically. The EIA using the purified 120 kDa protein had the highest specificity (92%) compared with the EIA using a whole cell sonicate of a single Helicobacter ***pylori*** strain as antigen helicobacter ***pylori*** strain as antigen (60.7%) and the immunoblot (90.2%). The sensitivity was 96%, 100% and 92%, respectively. Sera of three control patients reacted strongly in all three methods, indicating possible Helicobacter ***pylori*** infection with negative histological finding. The EIA using the 120 kDa protein as antigen was shown to be a specific and sensitive technique for the serodiagnosis of

Helicobacter ***pylori*** infections.

L11 ANSWER 30 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1992:524992 BIOSIS
DN BA94:133067
TI SERODIAGNOSIS OF HELICOBACTER- ***PYLORI*** INFECTIONS BY DETECTION OF IMMUNOGLOBULIN G ***ANTIBODIES*** USING AN IMMUNOBLOT TECHNIQUE AND ENZYME ***IMMUNOASSAY*** .
AU FAULDE M; SCHROEDER J P; SOBE D
CS ERNST-RODENWALDT-INSTITUT, FACHBEREICH II MED. MIKROBIOLOGIE, VIKTORIASTR. 11-13, 5400 KOBLENZ, GER.
SO EUR J CLIN MICROBIOL INFECT DIS, (1992) 11 (7), 589-594.
CODEN: EJCDEU. ISSN: 0934-9723.
FS BA; OLD
LA English
AB A transferable solid phase enzyme ***immunoassay*** (TSP-EIA) and an immunoblot technique were evaluated for the detection of IgG ***antibodies*** against Helicobacter ***pylori*** . Using the biopsy urease test as reference method, the sensitivity and specificity of the EIA were 96% and 100%, respectively. Immunoblot analyis was carried out by testing sera from patients with a positive urease test who suffered from type B ***gastritis*** , ***gastric*** and duodenal ulcers, and a negative control group. The immunoblotted Helicobacter ***pylori*** proteins showed reproducible immunoreactive bands at molecular weights of 130, 93, 75 and 67 kDa. The molecular weight protein fractions of Helicobacter ***pylori*** of 180 kDa and higher were found to be of minor immunological significance. Proteins of less than 60 kDa exhibited wide serum-specific variations in reactivity after immunostaining. No correlation between specific immunoblot patterns and clinical signs induced by Helicobacter ***pylori*** infection was observed.

L11 ANSWER 31 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1992:96603 BIOSIS
DN BA93:53153
TI EVALUATION OF A COMMERCIALLY AVAILABLE SECOND-GENERATION IMMUNOGLOBULIN G ENZYME ***IMMUNOASSAY*** FOR DETECTION OF HELICOBACTER- ***PYLORI*** INFECTION.
AU GOOSSENS H; GLUPCZYNSKI Y; BURETTE A; VAN DEN BORRE C; BUTZLER J-P
CS WORLD HEALTH ORGANIZATION COLLABORATING CENTRE ENTERIC CAMPYLOBACTER, ST-PIETERS UNIVERSITY HOSPITAL, HOOGSTRAAT 322, B-1000 BRUSSELS, BELGIUM.
SO J CLIN MICROBIOL, (1992) 30 (1), 176-180.
CODEN: JCMIDW. ISSN: 0095-1137.
FS BA; OLD
LA English
AB We evaluated a commercially available second-generation anti-H. ***pylori*** immunoglobulin G enzyme ***immunoassay*** (EIA) (Cobas Core Anti-Helicobacter ***pylori*** EIA; Roche S.A., Basel, Switzerland) for serodiagnosis of H. ***pylori*** infection. The results of the assay were assessed in relation to the results of bacterial culture, urease testing, and histological Giemsa stain of ***gastric*** biopsy specimens from 1,134 patients with a variety of symptoms relating to the upper gastrointestinal tract. H. ***pylori*** was detected in biopsy specimens from 660 (58.2%) patients: 6 had a normal mucosa, 123 had chronic ***gastritis*** only, and 531 were found to have chronic active ***gastritis*** by histology; endoscopy showed duodenal and

gastric ulcers in 137 and 64 patients of the last two groups, respectively. The test was evaluated with different age and ethnic groups. The prevalence, sensitivity, specificity, and positive and negative predictive values were, respectively, (i) for Belgian patients between 18 and 40 years old, 34, 93, 95, 91, and 96%; (ii) for Belgian patients more than 40 years old, 53, 96, 91, 93, and 95%; and (iii) for Mediterranean patients more than 17 years old, 87, 94, 70, 95, and 64%. All sera showing discordant ***immunoassay*** results compared with the results of histology and culture of biopsy specimens, as well as those with borderline ***immunoassay*** results, were tested further by immunoblotting. Among the EIA results considered false negative, we demonstrated an absence of seroconversion in 14 of 19 patients tested by immunoblotting. Among the EIA results considered false positive, immunoblotting showed the presence of specific ***antibodies*** in 28 of 37 patients tested. Among the borderline results obtained in the first assay with 22 patients' sera, a second assay showed positive results in 10 patients (8 were positive by immunoblotting) and negative reactions in 10 patients (9 were negative by immunoblotting), whereas 2 remained borderline. These data indicate that sera showing borderline ***immunoassay*** results must be tested again. In conclusion, this commercially available second-generation EIA, which is easy and quick to perform, was found highly reliable for the serodiagnosis of H. ***pylori*** infection.

L11 ANSWER 32 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1991:402414 BIOSIS
DN BR41:64259
TI EVALUATION OF AN ENZYME ***IMMUNOASSAY*** MEASURING SERUM ***ANTIBODY*** TO HELICOBACTER- ***PYLORI*** VS. WESTERN BLOT AND HISTOCHEMICAL UREASE TESTING.
AU PASKELL S; HOUGHTON R; VIGOREN E; THRESHER K
CS BAINBRIDGE LAB. INC., BAINBRIDGE, IS., WA 98110.
SO 91ST GENERAL MEETING OF THE AMERICAN SOCIETY FOR MICROBIOLOGY, DALLAS, TEXAS, USA, MAY 5-9, 1991. ABSTR GEN MEET AM SOC MICROBIOL. (1991) 91 (0), 343.
CODEN: AGMME8.
DT Conference
FS BR; OLD
LA English

L11 ANSWER 33 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1991:388110 BIOSIS
DN BA92:65425
TI A NOVEL ENZYME ***IMMUNOASSAY*** FOR SERODIAGNOSIS OF HELICOBACTER-***PYLORI*** INFECTION.
AU SUGIYAMA T; IMAI K; YOSHIDA H; TAKAYAMA Y; YABANA T; YOKOTA K; OGUMA K; YACHI A
CS DEP. INTERNAL MEDICINE, SAPPORO MEDICAL COLLEGE, S-1, W-16, CHUO-KU, SAPPORO 060, JPN.
SO GASTROENTEROLOGY, (1991) 101 (1), 77-83.
CODEN: GASTAB. ISSN: 0016-5085.
FS BA; OLD
LA English
AB Helicobacter ***pylori*** has recently been implicated as an etiologic agent of gastroduodenal disorders. Comparing the ***antibody*** to H.

pylori in the sera of patients with that of normal controls by Western blot analysis, a unique ***antibody*** was detected in the sera of patients, which reacted with the 25-kilodalton antigen of H. ***pylori*** . On the other hand, monoclonal ***antibody*** CP3 prepared in the authors' laboratory also recognized the 25-kilodalton antigen of H. ***pylori*** . Whether the serum ***antibody*** of the patient recognized the CP3 antigen purified by monoclonal ***antibody*** CP3 was then examined. Western blot analysis showed that the patient's serum reacted strongly with the affinity-purified CP3 antigen. Using monoclonal ***antibody*** CP3, an enzyme-linked immunosorbent assay to detect CP3 ***antibody*** in sera was established. In patients with chronic ***gastritis*** and ***gastric*** ulcers, the titer of CP3 ***antibody*** was significantly higher than in normal controls and correlated with the histological grade of antral ***gastritis*** . The detection of CP3 ***antibody*** in sera is useful in the diagnosis of chronic ***gastritis*** and ***gastric*** ulcer associated with H. ***pylori*** infection and also in evaluation of the grade ***gastritis*** .

L11 ANSWER 34 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1990:355466 BIOSIS
DN BA90:52045
TI INOCULATION OF BARRIER-BORN PIGS WITH HELICOBACTER- ***PYLORI*** A USEFUL ANIMAL MODEL FOR ***GASTRITIS*** TYPE B.
AU ENGSTRAND L; GUSTAVSSON S; JORGENSEN A; SCHWAN A; SCHEYNIUS A
CS DEP. CLINICAL BACTERIOLOGY, UNIVERSITY HOSPITAL, UPPSALA, SWED.
SO INFECT IMMUN, (1990) 58 (6), 1763-1768.
CODEN: INFIBR. ISSN: 0019-9567.
FS BA; OLD
LA English
AB At the age of 8 weeks, 15 barrier-born pigs, specific pathogen free, were inoculated intragastrically with suspension of 107 to 1010 CFU of Helicobacter ***pylori*** after treatment with omeprazole. The pigs were observed or up to 12 weeks, endoscopic biopsy specimens were taken, and serum samples were drawn. H. ***pylori*** was identified by routine culturing and by staining with an H. ***pylori*** -specific monoclonal ***antibody*** on cryostat sections of ***gastric*** biopsy specimens. In 11 of 15 inoculated pigs, H. ***pylori*** was detected throughout the observation period. In these infected pigs, there was an ***antibody*** response to H. ***pylori*** , as determined in serum by an enzyme ***immunoassay*** . Furthermore, the development of superficial, focal ***gastritis*** with infiltrates of mononuclear class II antigen-expressing lymphocytes was observed immunohistologically. H. ***pylori*** was never detected and an ***antibody*** response to H. ***pylori*** was not observed in two control pigs. The development of ***gastritis*** and the systemic ***antibody*** response to H. ***pylori*** support the usefulness of this animal model for studies of H. ***pylori*** -related human diseases.

L11 ANSWER 35 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1990:218383 BIOSIS
DN BA89:115673
TI ISOTYPE EVOLUTION IN THE FOLLOW-UP STUDY OF PATIENTS WITH CAMPYLOBACTER- ***PYLORI*** ASSOCIATED ***GASTRITIS*** .

AU GOBERT B; BENE M C; DE KORWIN J D; FAURE G
CS LAB. D'IMMUNOL., CHRU DE NANCY, BP 184, F-54500 VANDOEUVRE-LES-NANCY, FR.
SO GASTROENTEROL CLIN BIOL, (1989) 13 (11), 880-883.
CODEN: GCBIDC. ISSN: 0399-8320.
FS BA; OLD
LA English
AB Four sequential immuno-assays were performed from May to November 1988 to follow the levels of IgA, IgA and IgM to Campylobacter ***pylori*** in 16 infected patients with histologically proven ***gastritis*** , among which 12 received appropriate therapy. Histopathological examination of antral biopsies, bacteriological cultures and urease tests were performed on each occasion when serum was tested for ***antibodies*** to C. ***pylori*** . The detection and quantitative assessment of the various isotypes to this bacterium proved valuable to appreciate the response to therapy with, in case of success, a steady decrease of ***antibodies*** levels concomitant with clinical improvement.

L11 ANSWER 36 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1990:156704 BIOSIS
DN BA89:84122
TI VALUE OF SEROLOGY ELISA AND IMMUNOBLOTTING FOR THE DIAGNOSIS OF CAMPYLOBACTER- ***PYLORI*** INFECTION.
AU PENA A S; ENDTZ H P; OFFERHAUS G J A; HOOGENBOOM-VERDEGAAL A; VAN DUIJN W; DE VARGAS N; DEN HARTOG G; KREUNING J; VAN DER REYDEN J; ET AL
CS DEP. GASTROENTEROL., LEIDEN UNIV. HOSP., P.O. BOX 9600, NL-2300 RC LEIDEN, NETH.
SO DIGESTION, (1989 (1990)) 44 (3), 131-141.
CODEN: DIGEBW. ISSN: 0012-2823.
FS BA; OLD
LA English
AB Fifty-two unselected patients referred to for upper gastrointestinal endoscopy were evaluated in several ways to determine the presence of Campylobacter ***pylori*** . ***Antibodies*** against this microorganism were measured to assess the value of serology for the diagnosis of C. ***pylori*** infection. Five antral biopsy specimens were taken in each patient for culture and bacteriological determinations, histology [morphology and Warthin-Starry (WS) staining] and the urease test (2, 3 and 24 h). Serum ***antibodies*** against a sonicate of 6 strains of micoorganisms were assayed by enzyme-linked ***immunoassay*** (ELISA) and an immunoblotting technique. In 14 of the 52 patients the histology of the antrum was normal, 18 patients had chronic active ***gastritis*** and 20 had chronic ***gastritis*** without polymorphonuclear infiltration. In the group with normal histology, only 1 patient was positive for C. ***pylori*** with all methods, and 1 other subject was positive for IgG and 2 for IgA only with ELISA. In the group with chronic active ***gastritis*** , 14 were positive with all methods, 1 was negative by WS only and another was negative for IgA according to ELISA, WS and ***antibodies*** . Among the patients with chronic ***gastritis*** , 7 were positive and 7 negative with all tests; in the other 6 patients the results obtained with the various tests were divergent. Four serological tests were studied and validated against culture, WS and urease test which were considered to be the reference methods. The serological tests showed high sensitivity and specificity for the detection of C. ***pylori*** -associated active chronic ***gastritis*** of the antrum, and can therefore serve as noninvasive

methods to identify individuals with this condition.

L11 ANSWER 37 OF 184 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1989:336534 BIOSIS
DN BA88:39534
TI DIAGNOSTIC VALUE OF AN ***IMMUNOASSAY*** TO DETECT ANTI CAMPYLOBACTER-***PYLORI*** ***ANTIBODIES*** IN NON-ULCER DYSPEPSIA.
AU LOFFELD R J L F; STOBBERINGH E; FRENDRIG J A; VAN SPREEUWEL J P; ARENDS J W
CS DEP. INTERN. MED., UNIV. HOSPITAL MAASTRICHT, PO BOX 1918, 6201BX MAASTRICHT, NETH.
SO LANCET, (1989) 1 (8648), 1182-1185.
CODEN: LANCAO. ISSN: 0140-6736.
FS BA; OLD
LA English
AB An enzyme-linked immunosorbent assay (ELISA) for detection of IgG ***antibodies*** against Campylobacter ***pylori*** was used to examine sera from 70 patients with non-ulcer dyspepsia. 48 patients had C ***pylori*** associated ***gastritis*** according to culture or histology; mean optical density (OD) of the ELISA was significantly higher than that for the 22 patients with normal antral mucosa and absence of C ***pylori*** . Positive and negative predictive values for campylobacter-associated ***gastritis*** were 100% above OD 2.10 and below OD 1.00, respectively. Serology might replace endoscopy in the diagnosis of campylobacter-associated ***gastritis*** .

L11 ANSWER 38 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 2000:790743 CAPLUS
DN 133:319313
TI Method for assessing the risk of peptic ulcer, comprising the steps of determining quantitatively the concentrations of ***pepsinogen*** I (pgi) and ***gastrin*** -17 in a serum sample
IN Sipponen, Pentti; Harkonen, Matti; Suovaniemi, Osmo; Forsblom, Erik
PA Locus Genex Oy, Finland
SO PCT Int. Appl., 19 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000067035	A1	20001109	WO 2000-FI377	20000428
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
FI 9900992	A	20001031	FI 1999-992	19990430

PRAI FI 1999-992 19990430
AB The present invention concerns a method for assessing the risk of peptic ulcer by detg. the presence and topog. phenotype of ***gastritis*** in

an individual, by detg. quant. the ***pepsinogen*** I and ***gastrin*** -17 concns. in a serum sample from the said individual, selecting a method-specific ref. value and cut-off value for resp. analyte, assessing the topog. and phenotype of ***gastritis*** based on a comparison of the ***pepsinogen*** I and ***gastrin*** -17 concns. so detd. with their resp. method-specific ref. and cut-off values, and correlating the so assessed ***gastritis*** phenotype with the risk for peptic ulcer. Preferably also Helicobacter ***antibodies*** are detd. in the sample.

RE.CNT 3

RE

(1) Javier, P; European Journal of Gastroenterology & Hepatology 1999, V11, P189

(2) Locus Genex Oy; WO 9615456 A1 1996 CAPLUS

(3) Tseng-Shing, C; The American Journal of Gastroenterology 1994, V89(9), P1511

L11 ANSWER 39 OF 184 CAPLUS COPYRIGHT 2001 ACS

AN 2000:275448 CAPLUS

DN 133:280240

TI Serum reactivity to Helicobacter ***pylori*** antigens assessed by enzyme ***immunoassay*** and the immunoblot method in children

AU Wojda, Urszula; Dzierzanowska, Danuta; Witkowska-Vogtt, Ewa; Celinska-Cedro, Danuta

CS Department of Clinical Microbiology, Childrens Memorial Health Institute, Warsaw, Pol.

SO Cent.-Eur. J. Immunol. (1999), 24(4), 257-264
CODEN: CJIMFW; ISSN: 1426-3912

PB Polish Society for Immunology

DT Journal

LA English

AB H. ***pylori*** infection is the most common chronic bacterial disease in humans. It is the major cause of ***gastritis*** , peptic ulcer, ***gastric*** carcinoma, and MALT lymphoma. The aim here was to assess the usefulness of 2 serol. methods, EIA and immunoblot, for detecting IgG ***antibodies*** to H. ***pylori*** in children. A total of 382 serum samples were included in the study. Group 1 contained 282 serum samples from children with dyspeptic symptoms of the proximal alimentary tract. Group 2 contained serum samples from 100 children without any pathol. symptoms in the alimentary tract. The results of serol. studies revealed that as many as 181/282 (64.2%) of symptomatic children showed the presence of specific IgG ***antibodies*** to H. ***pylori*** antigens. In contrast, in the group of asymptomatic children, only 27/100 (27%) showed the presence of anti-H. ***pylori*** IgG ***antibodies*** in the serum. Specific ***antibodies*** against protein CagA (116 kDa) were detected much more frequently in children with ***gastritis*** 40/45 (88.8%) and in children with duodenitis 3/4 (75%) than in those with normal ***gastric*** mucosa 8/15 (53%). Detection of anti-H. ***pylori*** ***antibodies*** by the use of serol. methods appeared helpful in diagnosis of infections induced by the bacteria.

RE.CNT 24

RE

(2) Blaser, M; Cancer Res 1995, V55, P2111 CAPLUS

(4) Covacci, A; Proc Natl Acad Sci USA 1993, V90, P5791 CAPLUS

(10) Husson, M; J Clin Microbiol 1995, V33, P3300 CAPLUS
(12) Lutton, D; J Med Microbiol 1995, V42, P386 CAPLUS
(15) Meijer, B; J Clin Microbiol 1997, V35, P292 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 40 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 2000:195874 CAPLUS
DN 133:55175
TI Detection of Helicobacter ***pylori*** using surface plasmon resonance
AU Nishimura, Tomoaki; Hifumi, Emi; Uda, Taizo
CS School of Biosciences, Hiroshima Prefectural University, Shoubara, 727-0023, Japan
SO Chem. Sens. (1999), 15(Suppl. A, Proceedings of the 28th Chemical Sensor Symposium, 1999), 148-150
CODEN: KAGSEU
PB Denki Kagakkai Kagaku Sensa Kenkyukai
DT Journal
LA Japanese
AB Helicobacter ***pylori*** (H. pyroli) causes chronic ***gastritis*** and ***gastric*** ulcer. It is important to detect the H. pyroli to administer of antibiotics for patients. We have established a unique monoclonal ***antibody*** which has a specificity against H. pyroli urease. Moreover it hugely suppresses the enzymic activity of the urease. In this study, H. pyroli and its sonicated samples were detected by using a differential SPR. The detection limit by the SPR was 2 x 107 cell/mL to the sonicated sample, which showed much higher detection limit than unsonicated H. pyroli by 100 fold.

L11 ANSWER 41 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 2000:19479 CAPLUS
DN 132:63138
TI Helicobacter ***pylori*** antigens applicable to diagnosing Helicobacter ***pylori*** infection
IN Kondo, Isamu; Hoshina, Sadayori; Miki, Keizaburo
PA Japan
SO Jpn. Kokai Tokkyo Koho, 5 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2000002706	A2	20000107	JP 1998-183354	19980615

AB New Helicobacter ***pylori*** antigen or its fragments are provided so that they are applied to diagnosing Helicobacter ***pylori*** infection without giving a pain to a patient and distinguishing ulcer and duodenal ulcer from ***gastritis*** . These antigens are obtained from Helicobacter ***pylori*** present in ***gastric*** juice from ulcer patients or duodenal ulcer patients by the successive treatments with lysozyme and with N-acetylglucosaminidase, followed by electrophoresis. Their mol. wts. detd. by electrophoresis are .apprx.68 kDa, .apprx.78 kDa, .apprx.132 kDa and .apprx.140 kDa. The infection with Helicobacter ***pylori*** related to ulcer and duodenal ulcer, but not to ***gastritis*** , was diagnosed by detecting the specific IgA ***antibodies*** present in serum with these antigens.

L11 ANSWER 42 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1999:804109 CAPLUS
DN 132:292571
TI Effect of cagA status on the sensitivity of enzyme ***immunoassay*** in diagnosing Helicobacter ***pylori*** -infected children
AU Plebani, Mario; Guariso, Graziella; Fogar, Paola; Basso, Daniela; Gallo, Nicoletta; Zambon, Carlo Federico; Mozrzymas, Renata; Celadin, Marilena; Zacchello, Franco
CS Department of Laboratory Medicine, University Hospital of Padua, Italy
SO Helicobacter (1999), 4(4), 226-232
CODEN: HELIFL; ISSN: 1083-4389
PB Blackwell Science, Inc.
DT Journal
LA English
AB The authors sought to evaluate in symptomatic children the influence of the Helicobacter ***pylori*** genotype on ***gastritis*** , abdominal pain, and circulating anti-H. ***pylori*** IgG ***antibodies*** (anti-H. ***pylori*** IgG) or ***pepsinogen*** A (PGA) and C (PGC). Also, they assessed anti-H. ***pylori*** IgG, PGA, and PGC patterns in a large cohort of asymptomatic children. The infection was found in 33 of 183 symptomatic children; among the 20 H. ***pylori*** -pos. children for which the H. ***pylori*** genotype was available, cagA was present or absent in equal percentages. H. ***pylori*** infection was assocd. with more severe ***gastritis*** and higher serum levels of anti-H. ***pylori*** IgG and PGC but not with abdominal pain. In infected children, higher levels of anti-H. ***pylori*** IgG and the presence of abdominal pain were assocd. with infections caused by cagA-pos. strains. In the cohort of asymptomatic children, raised levels of anti-H. ***pylori*** IgG, PGA, and PGC were found in ~5% of the cases. Thus, infection with cagA-pos. H. ***pylori*** strains can be assocd. with increased frequency of reported abdominal pain and higher circulating levels of anti-H. ***pylori*** IgG. The serol. assessment of H. ***pylori*** IgG using H. ***pylori*** antigens contg. significant amts. of cagA protein may, therefore, underestimate the true prevalence of infection.
RE.CNT 38
RE
(2) Atherton, J; J Biol Chem 1995, V270, P17771 CAPLUS
(4) Blaser, M; Cancer Res 1995, V55, P2111 CAPLUS
(5) Blaser, M; Cancer Res 1995, V55, P562 CAPLUS
(8) Cover, T; Mol Microbiol 1996, V20, P241 CAPLUS
(19) Hunter, F; Dig Dis Sci 1993, V38, P2081 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 43 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1999:645846 CAPLUS
DN 132:219089
TI Anti-CagA reactivity in Helicobacter ***pylori*** -negative subjects: A comparison of three different methods
AU Fusconi, Marco; Vaira, Dino; Menegatti, Marcello; Farinelli, Silvia; Figura, Natale; Holton, John; Ricci, Chiara; Corinaldesi, Roberto; Miglioli, Mario
CS Servizio di Patologia Medica II, Istituto di Clinica Medica I, University of Bologna, Bologna, 40138, Italy

SO Dig. Dis. Sci. (1999), 44(8), 1691-1695
CODEN: DDSCDJ; ISSN: 0163-2116
PB Kluwer Academic/Plenum Publishers
DT Journal
LA English
AB Emerging evidence suggests that infection by CagA-pos. Helicobacter ***pylori*** strains is related to the development of more serious gastroduodenal diseases, thus conferring to the detn. of anti-CagA ***antibodies*** a relevant clin. significance in serol. screenings. The detection of anti-CagA positivity in sera neg. for anti-H. ***pylori*** ***antibodies*** raises the question of whether this apparently nonsense result is merely due to a false pos. reaction. To address this issue, we compared three different methods for the detection of anti-CagA ***antibodies*** . In all, 272 selected sera from patients with precisely defined H. ***pylori*** status (pos. or neg. concordance of five tests, ie, histol. by Giemsa in both antrum and corpus, rapid urease test, culture, [13C]urea breath test, IgG ELISA) were tested for anti-CagA reactivity by three different techniques (Western immunoblotting, ELISA, and recombinant immunoblotting assay). In order to assess the sensibility and specificity of each tests, we considered as "true" anti-CagA pos. sera those with two out of three pos. results. Sera from 70% of H. ***pylori*** -pos. patients and 10% from H. ***pylori*** -neg. patients turned out to be "true" positives for anti-CagA ***antibodies*** . The three methods showed similar excellent results, in terms of both sensitivity and specificity, always over 93%. It is confirmed that a proportion of patients with a neg. conventional serol. against H. ***pylori*** possess anti-CagA ***antibodies*** in their sera. In this paper we demonstrate that it can happen even in patients without any biol. signs of actual H. ***pylori*** infection. The possibility that this can be due to a false pos. lab. result is very likely ruled out by the accuracy of the three methods used. The clin. management of these patients needs further study on larger series.
RE.CNT 12
RE
(1) Blaser, M; Lancet 1997, V349, P1020 MEDLINE
(2) Censini, S; Proc Natl Acad Sci USA 1996, V93, P14648 CAPLUS
(3) Covacci, A; Proc Natl Acad Sci USA 1993, V90, P5791 CAPLUS
(4) Cover, T; J Clin Microbiol 1995, V33, P1496 MEDLINE
(11) Towbin, H; Proc Natl Acad Sci USA 1979, V76, P4350 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 44 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1998:784058 CAPLUS
DN 130:137953
TI Serological assessment of the early response to eradication therapy using an immunodominant outer membrane protein of Helicobacter ***pylori***
AU Nishizono, Akira; Gotoh, Takayuki; Fujioka, Toshio; Murakami, Kazunari; Kubota, Toshihiro; Nasu, Masaru; Watanabe, Makoto; Mifune, Kumato
CS Department of Infectious Diseases Control, Oita Medical University, Oita, 879-55, Japan
SO Clin. Diagn. Lab. Immunol. (1998), 5(6), 856-861
CODEN: CDIMEN; ISSN: 1071-412X
PB American Society for Microbiology
DT Journal

LA English .
AB Eradication of Helicobacter ***pylori*** infection cures ***gastritis*** and prevents recurrence of peptic ulcers. Endoscopy is usually used to evaluate the effectiveness of eradication therapy. We designed a new noninvasive assay system for the early evaluation of eradication of H. ***pylori*** infection in which a crude H. ***pylori*** outer membrane protein prepn. (HPOmp) is used as an antigen, and we detd. the sensitivity and specificity of the serol. assay system. Immunoblot anal. showed that anti-HPOmp ***antibodies*** reacted to a protein with a mol. mass of approx. 29 kDa. In those patients who responded to therapy, the anti-HPOmp IgG (IgG) titers measured by ELISA (ELISA) at 1 mo after the end of therapy were significantly lower than those before treatment (34.8% redn.; $P < 0.001$), and the posttreatment redn. in the ***antibody*** titer was significantly greater than that of the titer measured with a com. available anti-H. ***pylori*** IgG ELISA (34.8% vs. 16.1%; $P < 0.001$). When a 25% redn. of anti-HPOmp IgG titer at 1 mo after the end of treatment was taken as the cutoff value for H. ***pylori*** eradication, the sensitivity and specificity of our new assay were 75% (51 of 68 treatment responders) and 96% (22 of 23 nonresponders), resp. Our results indicate that the novel serol. test with HPOmp might be a clin. useful tool for assessment of eradication of H. ***pylori*** .
RE.CNT 15
RE
(2) Cullen, D; Lancet 1992, V340, P1161 MEDLINE
(4) Evans, D; Infect Immun 1989, V57, P664 CAPLUS
(5) Graham, D; Ann Intern Med 1992, V116, P705 MEDLINE
(10) Nishizono, A; J Clin Microbiol 1993, V31, P1173 CAPLUS
(15) Tomb, J; Science 1997, V388, P539 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 45 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1998:747526 CAPLUS
DN 130:51328
TI Reagent for detecting anti-Helicobacter ***pylori*** ***antibody***
IN Nishizono, Akira; Fujioka, Toshio; Mifune, Kumato; Watanabe, Makoto; Azumi, Junichi
PA Fujirebio, Inc., Japan
SO Jpn. Kokai Tokkyo Koho, 5 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 10307142	A2	19981117	JP 1997-130552	19970506

AB The disclosed reagent comprises Helicobacter ***pylori*** -derived extracellular membrane 10.apprx.100 kDa mol. The 10.apprx.100 kDa Helicobacter antigen is useful for ***immunoassay*** in detecting Helicobacter ***pylori*** -specific ***antibody*** and for diagnosing Helicobacter infection, e.g. ***gastritis*** .

L11 ANSWER 46 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1998:535180 CAPLUS
DN 129:215706

TI Diagnostic agent for digestive tract diseases derived from Helicobacter infection
IN Yokota, Shinichi; Amano, Kenichi
PA Sumitomo Pharmaceuticals Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 5 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 10218900	A2	19980818	JP 1997-32758	19970131

AB Disclosed is purified and immobilized Helicobacter ***pylori*** lipopolysaccharide for diagnosing the seriousness of digestive tract diseases derived from Helicobacter infection. Serum samples derived from patients with chronic ***gastritis*** , ***gastric*** ulcer and ***gastric*** cancer were tested with microplate contg. immobilized lipopolysaccharide for the presence of anti-Helicobacter lipopolysaccharide ***antibody*** .

L11 ANSWER 47 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1998:197684 CAPLUS
DN 128:228250
TI Adhesins and adhesive proteins from Helicobacter ***pylori*** and their diagnostic and therapeutic uses
IN Ho, Bow
PA Cortecs International Limited, Australia; Chapman, Paul William; Ho, Bow
SO PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9812562	A1	19980326	WO 1997-GB2554	19970922
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE; GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9743116	A1	19980414	AU 1997-43116	19970922
ZA 9708513	A	19990323	ZA 1997-8513	19970922

PRAI GB 1996-19694 19960920
GB 1996-22846 19961101
WO 1997-GB2554 19970922

AB The present invention relates to novel methods of diagnosing Helicobacter ***pylori*** infection in a subject based on the immunol. interactions of H. ***pylori*** adhesive proteins and adhesin in particular. In addn., the invention relates to methods for distinguishing between different ***gastric*** disease states caused by H. ***pylori*** including kits for use in such methods. Furthermore, the invention relates to the use of adhesin proteins in the prodn. of vaccines.

L11 ANSWER 48 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1997:776267 CAPLUS
DN 128:58315
TI Cloning, detection, and, biological activity of Helicobacter ***pylori*** ***gastric*** acid secretion inhibitory factor 1
IN Cave, David R.; Huang, Lili; Hoffman, James S.
PA St. Elizabeth's Hospital of Boston, USA
SO PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9744464	A1	19971127	WO 1997-US8018	19970514

W: CA, JP
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRAI US 1996-648986 19960517
AB The gene encoding acid inhibitory factor 1 (AIF-1) produced by Helicobacter ***pylori*** bacteria, was cloned and sequenced in its entirety and expressed in Escherichia coli. On the basis of a partial N-terminal amino acid sequence, a degenerate oligonucleotide was used to probe. an H. ***pylori*** gene library. An ***antibody*** to the N-terminal peptide was made, and an AIF-1 ELISA is described. AIF-1 is characterized as having a mol. mass of .apprx.90 kDa, an isoelec. point of 7.3, is inactivated by boiling temps. and the enzyme pronase. Its DNA sequence encodes a deduced amino acid sequence of 381 amino acids. Purified AIF-1 inhibits acid prodn. by ***gastric*** parietal cells.

L11 ANSWER 49 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1996:731124 CAPLUS
DN 126:70729
TI Immunomagnetic bead enrichment and PCR for detection of Helicobacter ***pylori*** in human stools
AU Nilsson, Hans-Olof; Aleljung, Paer; Nilsson, Ingrid; Tyszkiewicz, Tadeusz; Wadstroem, Torkel
CS Department of Medical Microbiology, University of Lund, Soelvegatan 23, S-223 62, Lund, Swed.
SO J. Microbiol. Methods (1996), 27(1), 73-79
CODEN: JMIMDQ; ISSN: 0167-7012
PB Elsevier
DT Journal
LA English
AB An immunomagnetic bead-based polymerase chain reaction assay (IMS-PCR) was developed for the detection of Helicobacter ***pylori*** in exptl. inoculated human stools and human clin. stool samples. Magnetic beads coated with anti-H. ***pylori*** rabbit ***antibodies*** were used for enrichment and concn. of H. ***pylori*** from fecal samples. Taq polymerase inhibitors, found in human feces, are efficiently removed by the immunomagnetic sepn. (IMS) and subsequent washing of the magnetic beads. Conditions of the assay were developed and optimized with feces from a healthy, H. ***pylori*** seroneg., individual. Feces was inoculated with serial dilns. of either the spiral or the coccoid form of H. ***pylori*** . These 2 morphol. forms could be detected at similar

concns. when inoculated in feces using an optimized IMS-PCR method. In 1 g of feces less than 2.5 .times. 104 H. ***pylori*** cells were detected as measured with 2 sep. sets of PCR-primers, based on a urease A subunit gene sequence and a gene sequence encoding a 26-kDa surface protein of H. ***pylori*** . Previously, no report has shown a sensitivity below 106 H. ***pylori*** in feces PCR. Preliminary anal. of stool samples from 17 patients with symptoms of ***gastritis*** and esophagitis by IMS-PCR showed a good correlation with EIA-anal. of H. ***pylori*** serum- ***antibodies*** from these patients. The results indicate that H. ***pylori*** cells are shed in feces of infected patients and that immunomagnetic bead PCR might be an appropriate method for clin. diagnosis and studies involving immunoprophylaxis, antibiotic treatment, as well as vaccine candidates.

L11 ANSWER 50 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1996:453992 CAPLUS
DN 125:108455
TI Novel ELISA kits for serum pepsinogens using polyclonal ***antibody*** : Correlation with conventional RIA kits and clinical significance
AU Matsukawa, Yoshihiro; Nishinarita, Susumu; Horie, Takashi; Matsumoto, Kyoici; Ishikawa, Masazumi; Hirooka, Tatsuo; Aoki, Takahito; Iwai, Chikara; Kurosaka, Hanzo
CS School Medicine, Nihon University, Itabashi, 173, Japan
SO Nihon Univ. J. Med. (1996), 38(3), 141-148
CODEN: NUMDAE; ISSN: 0546-0352
DT Journal
LA English
AB We established novel ELISA kits for estg. serum ***pepsinogen*** I and ***pepsinogen*** II using polyclonal ***antibodies*** against pepsinogens. The correlations with results obtained with conventional kits for ***pepsinogen*** and the clin. significance of the novel kits were also evaluated. We measured the serum ***pepsinogen*** levels of endoscopically normal subjects and patients with gastroduodenal diseases employing both kits. The results for the serum ***pepsinogen*** levels measured with the ELISA kits correlated well with those measured with the conventional RIA kits (r=0.98 for both pepsinogens I and II). Using the ELISA kits, the serum ***pepsinogen*** II levels were found to be elevated in patients with ***gastric*** and duodenal ulcer as compared to those of endoscopically normal subjects (and 0.05). Concerning the serum ***pepsinogen*** I, patients with ***gastric*** polyp, chronic atrophic ***gastritis*** , and intestinal metaplasia manifested lower levels than did normal subjects (0.01, and 0.01). The ratio of serum ***pepsinogen*** I to ***pepsinogen*** II were lower in patients with ***gastric*** cancer, ***gastric*** ulcer, ***gastric*** polyp, chronic atrophic ***gastritis*** , and intestinal metaplasia as compared to those of in normal subjects (for ***gastric*** polyp, and 0.01 for the others). The ELISA kits were beneficial from the view point of radiohazard problems, and appeared to be useful tools for evaluating the condition of the gastroduodenal mucosa.

L11 ANSWER 51 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1996:188384 CAPLUS
DN 125:55676
TI Helicobacter ***pylori*** infection. Part 2. Comparison of various serological tests

AU Haeckel, R.; Haeckel, Hella; Dirks, H.; Koessling, F.
CS Inst. Laboratoriumsmed., Zentralkrankenhaus St. Juergenstrasse, Bremen, D-28205, Germany
SO Laboratoriumsmedizin (1996), 20(2), 87-91
CODEN: LABOD3; ISSN: 0342-3026
DT Journal
LA German
AB IgG ***antibodies*** against H. ***pylori*** were detd. by the qual. Helisal Rapid Blood Test, and by 3 quant. tests (Enzygnost anti-Helicobacter ***pylori*** , Helicobacter ***pylori*** ***Antibody*** test, and G.A.P. Test IgG). The estn. was divided in 2 groups (with and without acute ***gastritis*** caused by H. ***pylori***). The quant. ***antibody*** test gave the best results with 91% sensitivity and 96% specificity.

L11 ANSWER 52 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1994:215316 CAPLUS
DN 120:215316
TI Rapid in vitro test and kit for Helicobacter ***pylori*** in saliva
IN Cripps, Allan W.; Stiel, Daniel; Witt, Campbell S.; Clancy, Robert L.
PA Auspharm International Ltd., Australia
SO Pat. Specif. (Aust.), 30 pp.
CODEN: ALXXAP
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI AU 644121	B2	19931202	AU 1990-67676	19901203
AU 9067676	A1	19910606		
PRAI AU 1989-7718		19891204		

AB A method for detecting contemporary infection by H. ***pylori*** in a mammal comprises contacting a mucous secretion from the mammal with an antigen component from H. ***pylori*** for a time and under conditions sufficient for an IgG ***antibody*** in the mucous secretion specific to the antigen component to form a complex and then subjecting the complex to a detecting means. The antigen component is immobilized onto a solid support (e.g., nitrocellulose membrane, glass, polymer). The antigen component of H. ***pylori*** comprises whole cell ext. and/or one or more isolated components thereof. The isolated component comprises a protein, a lipopolysaccharide, a polysaccharide, a lipid or any combination thereof. A kit is also disclosed. IgG ***antibodies*** to H. ***pylori*** were detd. in saliva samples by ELISA; the ***antibody*** levels were directly related to the level of H. ***pylori*** infection as indicated by the degree of ***gastric*** inflammation.

L11 ANSWER 53 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1990:156555 CAPLUS
DN 112:156555
TI Detection of Campylobacter ***pylori*** urease ***antibodies*** and reagent therefor
IN Dent, Julie Claire
PA UK
SO PCT Int. Appl., 21 pp.

CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 8909407	A1	19891005	WO 1989-GB104	19890206
W: AT, AU, BB, BG, BR, CH, DE, DK, FI, GB, HU, JP, KP, KR, LK, LU, MC, MG, MW, NL, NO, RO, SD, SE, SU, US				
RW: AT, BE, BJ, CF, CG, CH, CM, DE, FR, GA, GB, IT, LU, ML, MR, NL, SE, SN, TD, TG				
AU 8930547	A1	19891016	AU 1989-30547	19890206

PRAI GB 1988-6831 19880323
GB 1988-18531 19880804
WO 1989-GB104 19890206

AB A reagent for use in the diagnosis of C. ***pylori*** infections comprises C. ***pylori*** urease attached to a solid surface, e.g. a microplate or latex beads. The reagent is used to detect ***antibodies*** to C. ***pylori*** urease in a serum sample. A test kit for use in diagnosis of C. ***pylori*** infections is provided. Thus, a std. ELISA protocol employing the above reagent was used to test for the presence of ***antibodies*** to C. ***pylori*** urease in 202 patients receiving gastroscopy. There was a high correlation between the presence of proven ***gastritis*** and ***antibody*** to C. ***pylori*** urease. A very high discrimination was found between C. ***pylori*** neg. and pos. patients on microbiol. testing when the urease ELISA was used in testing serum. Eighteen sera from C. ***pylori*** -pos. patients which had been neg. on an ELISA using a sol. antigen gave high ***antibody*** titers with the urease ELISA.

L11 ANSWER 54 OF 184 CAPLUS COPYRIGHT 2001 ACS
AN 1990:117176 CAPLUS
DN 112:117176
TI Antigenic compositions of Campylobacter ***pylori*** and methods for their production and diagnostic use
IN Blaser, Martin J.
PA USA
SO Eur. Pat. Appl., 23 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 329570	A2	19890823	EP 1989-400464	19890217
EP 329570	A3	19910522		
EP 329570	B1	19970502		
R: AT, BE, CH, DE, ES, FR, GB, GR, IT, LI, LU, NL, SE				
US 5459041	A	19951017	US 1988-158003	19880218
CA 1339067	A1	19970729	CA 1989-591107	19890215
JP 02035096	A2	19900205	JP 1989-39167	19890217
JP 2738947	B2	19980408		
AT 152524	E	19970515	AT 1989-400464	19890217
ES 2100850	T3	19970701	ES 1989-400464	19890217

PRAI US 1988-158003 19880218

AB ***Antibodies*** to C. ***pylori*** are detected using an antigenic compn. comprising fragments (esp. of the flagella) of 63, 57, 45, and/or 31 kilodaltons for diagnosis of ***gastritis*** or peptic ulcer disease. Levels of IgG, IgA, and IgM specific for C. ***pylori*** were detd. in blood serum samples, taken 8-581 days after ingestion of C. ***pylori*** , by ELISA using polystyrene well-immobilized antigens of 5 strains of C. ***pylori*** . Saroconversion in the IgA and IgG classes occurred between days 60 and 431 following challenge. For IgM there was a nearly 4-fold increase in optical d. between day 8 and 22 after challenge and a gradual decline afterwards. For diagnostic purposes, pos. threshold was 0.910, 0.470, and 2.6 optical d. units for IgG and IgA in blood sera and for IgG in urine, resp.

L11 ANSWER 55 OF 184 CAPLUS COPYRIGHT 2001 ACS

AN 1988:92717 CAPLUS

DN 108:92717

TI Enzyme-linked immunosorbent assay of proton-potassium ***ATPase*** , the parietal cell antigen

AU Karlsson, F. A.; Burman, Pia; Loof, L.; Olsson, M.; Scheynius, Annika; Mardh, S.

CS Dep. Med. Physiol. Chem., Univ. Uppsala, Uppsala, S-751-85, Swed.

SO Clin. Exp. Immunol. (1987), 70(3), 604-10
CODEN: CEXIAL; ISSN: 0009-9104

DT Journal

LA English

AB Vesicular membranes, purified from porcine ***gastric*** mucosa and rich in H+, K+- ***ATPase*** , were used to establish an enzyme-linked immunosorbent assay (ELISA) for detns. of parietal cell autoantibodies. Results obtained with the ELISA correlated well with std. immunofluorescence detns. of parietal cell ***antibodies*** based on frozen sections of rat stomach. The ELISA however was .apprx.10-fold more sensitive than the immunofluorescence method and had high specificity. Intra- and interassay coeffs. of variation, detd. with a patient sera of av. positivity, were 5.5% and 18%, resp. The ELISA detected ***antibody*** binding in 23 of 26 sera from patients with known autoimmune atrophic ***gastritis*** , in 5 of 25 sera with autoimmune thyroiditis, in 5 of 20 sera from patients with Graves' disease, in 3 of 20 sera from patients with atoxic nodular goiter, in 6 of 20 sera of patients with primary biliary cirrhosis, in 2 of 20 sera of patients with active duodenal ulcer, in 2 of 20 sera with detectable antinuclear ***antibodies*** , and in 1 of 20 sera with detectable rheumatoid factor. Data detd. by an ELISA based on a ***gastric*** vesicular membrane prepn. of human origin correlated well to those obtained by the std. ELISA based on porcine membrane material. The assay should be well suited for routine detns. of parietal cell ***antibodies*** in investigations of autoimmune ***gastritis*** and multiple organ autoimmune endocrinopathies.

L11 ANSWER 56 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

AN 2000332591 EMBASE

TI Analysis of Helicobacter ***pylori*** binding site on HEp-2 cells and three cell lines from human ***gastric*** carcinoma.

AU Nishihara K.; Nozawa Y.; Nomura S.; Kitazato K.; Miyake H.

CS K. Nishihara, Pharmacology Research Lab., Tokushima Research Center, Taiho

Pharmaceutical Co. Ltd., 224-2, Ebisuno, Hiraishi, Tokushima 771-0132, Japan
SO Fundamental and Clinical Pharmacology, (1999) 13/5 (555-561).
Refs: 36
ISSN: 0767-3981 CODEN: FCPHEZ
CY France
DT Journal; Article
FS 004 Microbiology
016 Cancer
048 Gastroenterology
LA English
SL English
AB Helicobacter ***pylori*** (H. ***pylori***) is a pathogen responsible for chronic ***gastritis*** and peptic ulcer diseases. It colonises the ***gastric*** mucus layer and adheres to the ***gastric*** epithelial cell surface. As this adherence is the first step of infection, it is important to study the adherence mechanism. The aim of this study was to analyse the specific binding assay of H. ***pylori*** to HEp-2 cells and three ***gastric*** phenotype cell lines, AGS, MKN-45 and AZ-521. H. ***pylori*** NCTC 11637 grown on agar plates was harvested and used in experiments. H. ***pylori*** was inoculated to pre-cultured cell monolayers. Adhered bacteria were labelled with an anti-H. ***pylori*** ***antibody*** and an FITC-conjugated secondary ***antibody*** and quantified by using a fluorescent plate reader. Microbial adherence to HEp-2 cells increased with incubation time and incubated concentration of H. ***pylori*** . No further increase was obtained with four or more hours of incubation or with a concentration of 4 x 107 bacteria/well or more. Scatchard analysis revealed a linear plot and the Bmax value was 88.3. Similar adherence patterns were obtained when AGS, AZ-521 and MKN-45 cells were used for adherence assays, but they had a lower binding affinity than HEp-2 cells and AZ-521. MKN-45 cells had less receptors than HEp-2 and AGS cells. In conclusion, H. ***pylori***. adhered to the cell surface could be quantified by this assay method. H. ***pylori*** adhesion to cell surfaces has a single population of binding site and one type of binding site on HEp-2, AGS, AZ-521 and MKN-45 cells. (C) 1999 Editions scientifiques et medicales Elsevier SAS.

L11 ANSWER 57 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 2000301502 EMBASE
TI The clinical role of stool test (HpSA) in noninvasive diagnosis of Helicobacter ***pylori*** infection.
AU Vaira D.; Ricci C.; Acciardi C.; Gatta L.; Berardi S.; Miglioli M.
CS Dr. D. Vaira, 1st Medical Clinic, University of Bologna, Nuove Patologie Policlin. S. Orsola, Bologna, Italy. vairadin@med.unibo.it
SO Turkish Journal of Gastroenterology, (2000) 11/2 (97-102).
Refs: 30
ISSN: 1300-4948 CODEN: TJGAF3
CY Turkey
DT Journal; Article
FS 004 Microbiology
006 Internal Medicine
026 Immunology, Serology and Transplantation
048 Gastroenterology
LA English
SL English

AB Helicobacter ***pylori*** (H. ***pylori***) causes a chronic ***gastric*** infection, which is usually life-long and many epidemiological studies have shown that this is probably one of the most common bacterial infections throughout the world, involving 50% of the population in developed countries and up to 80-90% of the population in developing regions. It is therefore clean that nowadays the diagnosis of H. ***pylori*** infection today represents, at the very least, a key step in the management of many of the patients referred to the gastroenterologist. Due to the widerange and relevance of pathologies possibly related to infection (including malignancies), it also harbours the potential to become a major health problem. Up to now, there were only two widely available non-invasive methods: 1) 13C or 14C labelled urea breath test and 2) serology (which is based on the detection of a specific anti-H. ***pylori*** immune response, mostly by IgG ***antibodies*** , in patient's serum). Over the last few years H. ***pylori*** has been detected in the culture of stool samples but viable organisms are present only in a small percentage of cases. Despite the difficulties encountered in culture from stool samples, the fact that the organism was present at all raised the possibility of developing a new non-invasive diagnostic test based on the detection of bacterial antigen in stool. Over the last two years an enzymatic ***immunoassay*** (EIA), which detects the presence of H. ***pylori*** antigen in stool specimen has become available (HpSA(TM)- H. ***pylori*** Stool Antigen Meridian Diagnostics Inc., Cincinnati USA) and begun to be used in clinical practice to evaluate its performance compared to that of other currently available diagnostic tests. The HpSA test has recently received approval from the United States Food and Drugs Administration (FDA) for two indications for use: 1) diagnosis of H. ***pylori*** infection in adult symptomatic patients and 2) monitoring response and post-therapy in adult patients. The test utilises polyclonal anti-H. ***pylori*** capture ***antibody*** absorbed in microwells. It is clear that such a test, which detects bacterial antigen in an actual ongoing infection, is theoretically useful not only for screening, but also as an early predictor of successful treatment. This review will briefly consider the currently available evidence supporting a possible role for this non-invasive diagnostic test.

L11 ANSWER 58 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 2000258461 EMBASE
TI Noninvasive tests as a substitute for histology in the diagnosis of Helicobacter ***pylori*** infection.
AU Hahn M.; Fennerty M.B.; Corless C.L.; Magaret N.; Lieberman D.A.; Faigel D.O.
CS Dr. D.O. Faigel, Division of Gastroenterology, Portland VA Medical Center (P3GI), 3710 SW US Veterans Hospital Rd., Portland, OR 97201, United States
SO Gastrointestinal Endoscopy, (2000) 52/1 (20-26).
Refs: 25
ISSN: 0016-5107 CODEN: GAENBQ
CY United States
DT Journal; Article
FS 004 Microbiology
027 Biophysics, Bioengineering and Medical Instrumentation
036 Health Policy, Economics and Management
048 Gastroenterology

LA English
SL English
AB Background: Rapid urease tests for Helicobacter ***pylori*** have a sensitivity of 80% to 90%. Therefore histologic examination of ***gastric*** biopsies is recommended as a 'backup' diagnostic test in rapid urease test- negative patients. However, noninvasive tests (urea breath test, serology, whole blood ***antibody*** tests) may provide a more rapid diagnosis and be less expensive but offer similar accuracy. Methods: Sixty-seven patients (no prior treatment for H ***pylori*** , no proton pump inhibitors, antibiotics, or bismuth within 4 weeks) undergoing endoscopy for evaluation of dyspepsia symptoms and testing rapid urease test-negative by antral biopsy were enrolled. All had the following tests: ***gastric*** biopsies (2 antral, 1 fundus; H and E and Alcian Yellow stain) examined for ***gastritis*** and H ***pylori*** ; 13C-UBT; capillary blood for whole blood rapid ***antibody*** tests: FlexSure HP, QuickVue, AccuStat, and Stat-Simple ***Pylori*** ; serum for FlexSure HP; HM-CAP enzyme-linked ***immunoassay*** . H ***pylori*** infection was diagnosed (reference standard) if chronic ***gastritis*** was present on histology and at least 2 of the 3 following tests were positive: urea breath test, H ***pylori*** organisms unequivocally demonstrated in biopsies on special stain, and/or enzyme-linked ***immunoassay*** . The test and treatment costs per patient were calculated. Results: Of 67 patients with a negative rapid urease test, 4 were positive for H ***pylori*** . None had active peptic ulcer disease. Histology only identified I patient with organisms visible on special stain. Using chronic active ***gastritis*** (neutrophilic and mononuclear infiltrate) as a diagnostic criterion for H ***pylori*** , 6 patients would have been judged positive. However, only 2 of these were truly positive by the reference standard (positive predictive value 33%). Negative predictive value for presence of organisms and chronic active ***gastritis*** was 95% and 97%, respectively. All of the noninvasive tests identified all 4 truly positive patients correctly. Urea breath test and FlexSure whole blood assay yielded a substantial number of false-positive results (positive predictive value 31% and 36%, respectively); positive predictive value for the other tests ranged from 50% to 80%. All tests except histology had a negative predictive value of 100%. Histology was the most costly test ($p < 0.001$ compared with all other tests), followed by urea breath test and HM-CAP serology ($p < 0.001$ compared with all rapid ***antibody*** tests). Conclusions: Whole blood or serum ***antibody*** testing is a rapid, accurate, and cost- effective means for establishing H ***pylori*** status in rapid urease test- negative patients. Whole blood or serology rapid ***antibody*** testing should substitute for histology when the patient has not been previously treated for H ***pylori*** .

L11 ANSWER 59 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 2000206472 EMBASE
TI Telomerase expression, Hp infection and ***gastric*** mucosal carcinogenesis.
CS Dr. X.X. He, Department of Gastroenterology, First Affiliated Hospital, Sun Yat-sen Univ. of Med. Sciences, 58 Zhongshanerlu, Guangzhou 510080, Guangdong Province. hexingxiang@263.net
SO World Chinese Journal of Digestology, (2000) 8/5 (505-508).
Refs: 11
ISSN: 1009-3079 CODEN: SHXZF2

CY China
DT Journal; Article
FS 004 Microbiology
005 General Pathology and Pathological Anatomy
016 Cancer
048 Gastroenterology
LA Chinese
SL English; Chinese
AB AIM: To analyze telomerase activity and whether it is implicated in Hp infection as well as the relationship among telomerase expression, Hp infection and ***gastric*** mucosal carcinogenesis. METHODS: Telomerase activity was detected by TRAP in normal ***gastric*** mucosa, precancerous lesions and ***gastric*** carcinoma. Serum Hp-CagA-IgG ***antibody*** was determined by EIA in Hp infected patients. The relationship between telomerase activity and Hp-CagA- IgG ***antibody*** was studied by match method using 22 pairs of Hp positive patients including corresponding non-neoplastic ***gastric*** mucosa of ***gastric*** carcinoma and chronic superficial ***gastritis*** (CSG) mucosa. RESULTS: All normal ***gastric*** mucosa and CSG were telomerase negative. The positive rates of telomerase activity of grade 0 (n = 20), 1 (n = 40) and 2 (n = 8) intestinal metaplasia (IM) are 0%, 25% and 38%, respectively. In 68 chronic atrophic ***gastritis*** (CAG), 16 of 18 ***gastric*** carcinoma showed telomerase activity, the positive rate was the highest 89% in all the biopsy specimens. Thirty-nine of 45 tumors had telomerase activity (89%). The positive rates of telomerase activity of IM grade 0 (n = 15), 1 (n = 22) and 2 (n = 8) were 0%, 32% and 100%, respectively in corresponding nontumorous tissues. The incidence of IM grade 2 or tumor specimens was significantly higher than that in IM grade 0 or 1 ($P<0.01$). The Hp positive rates at normal ***gastric*** (n = 10), CSG (n = 46), CAG IM grade 0 (n = 20), 1 (n = 40) and 2 (n = 8) were 0%, 52%, 60%, 70% and 75%, respectively. Hp infection increased as the grade of IM advanced, in parallel with telomerase expression in the CAG. Hp-CagA-IgG ***antibody*** in CSG patients was significantly lower than that in patients with ***gastric*** carcinoma ($P<0.01$). All of 22 Hp positive ***gastric*** carcinoma were CagA+ strains (100.%). Twelve (55%) of the 22 corresponding nontumorous ***gastric*** mucosa had positive telomerase activity. In contrast, only 8 of 22 Hp positive CSG were infected CagA+ strains (36%); and all of the 22 ***gastric*** mucosa showed negative telomerase. CONCLUSION: The normal and CSG mucosa express no telomerase activity. Telomerase activity expression increases as the grade of IM advanced in CAG. Telomerase activity of ***gastric*** carcinoma is the highest (>88%) in all the ***gastric*** mucosa. The degree of Hp infection increases in parallel with telomerase positively in corresponding non-cancerous mucosa of ***gastric*** cancer. Infected Hp is usually CagA+ strain in cancer, whereas infected Hp is usually CagA- strain in CSG. Thus, telomerase activity may serve as a powerful tool for an early ***gastric*** carcinoma diagnosis. CagA+ Hp infection may be a strong trigger for telomerase reactivation.

L11 ANSWER 60 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 2000144389 EMBASE
TI Diagnosis of Helicobacter ***pylori*** infection in patients with atrophic ***gastritis*** : Comparison of histology, 13C-urea breath test, and serology.

AU Kokkola A.; Rautelin H.; Puolakkainen P.; Sipponen P.; Farkkila M.; Haapiainen R.; Kosunen T.U.
CS Dr. P. Puolakkainen, Second Dept. of Surgery, Helsinki University, Central Hospital, Haartmaninkatu 4, FIN-00290 Helsinki, Finland
SO Scandinavian Journal of Gastroenterology, (2000) 35/2 (138-141).
Refs: 27
ISSN: 0036-5521 CODEN: SJGRA4
CY Norway
DT Journal; Article
FS 004 Microbiology
005 General Pathology and Pathological Anatomy
016 Cancer
026 Immunology, Serology and Transplantation
048 Gastroenterology
LA English
SL English
AB Background: Atrophic ***gastritis*** , a risk factor for ***gastric*** cancer, is a late consequence of Helicobacter ***pylori*** infection in approximately one-third of the infected patients. It has been suggested that ***gastric*** cancer would develop less frequently if H. ***pylori*** were eradicated. However, the prevalence of H. ***pylori*** infection may be underestimated in patients with atrophic ***gastritis*** and intestinal metaplasia if only biopsy-based diagnostic methods are used. Methods: We compared histology, 13C-urea breath test (13C-UBT), and serology in H. ***pylori*** diagnostics in 50 male patients with atrophic corpus ***gastritis*** . Results: H. ***pylori*** was detected in 15 (30%)patients by histology and in 14 (28%) by 13C-UBT, whereas increased serum ***antibody*** levels indicating H. ***pylori*** infection were found in 41 (82%) patients ($P < 0.0001$ between serology and both histology and 13C-UBT). H. ***pylori*** infection was associated with atrophic corpus ***gastritis*** in 84% of the present patients (in one patient with normal ***antibody*** titres H. ***pylori*** was defined histologically). Conclusions: H. ***pylori*** infection would have been missed in most patients with atrophic ***gastritis*** without the analysis of H. ***pylori*** ***antibodies*** . Therefore, in patients with atrophic ***gastritis*** , the use of serology is encouraged in diagnosing H. ***pylori*** infection.

L11 ANSWER 61 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 1999423461 EMBASE
TI Use of serology, the urease test and histology in diagnosis of Helicobacter ***pylori*** infection in symptomatic and asymptomatic Indians.
AU Kang G.; Rajan D.P.; Patra S.; Chacko A.; Mathan M.M.
CS Dr. G. Kang, Department Gastrointestinal Sciences, Christian Medical College Hospital, Vellore 632004, India
SO Indian Journal of Medical Research, (1999) 110/SEP. (86-90).
Refs: 17
ISSN: 0971-5916 CODEN: IMIREV
CY India
DT Journal; Article
FS 004 Microbiology
048 Gastroenterology
LA English

SL English
AB Age-specific prevalence of IgA and IgG ***antibodies*** in 714 subjects without gastrointestinal complaints aged 6 months to 90 yr was measured by an enzyme linked ***immunoassay*** using an acid-glycine extract of H. ***pylori*** as the antigen. The urease test and histology were used for the diagnosis of H. ***pylori*** infection in 83 subjects with a clinical diagnosis of dyspepsia, and these results were compared with measurement of IgG, IgA and IgM ***antibodies*** . The age specific prevalence of IgG and IgA ***antibodies*** respectively was 57 and 43 per cent for subjects aged 6 months to 4 yr and showed an increase with age to a maximum of 90 per cent for IgG in subjects > 60 yr of age and to 87 per cent for IgA in subjects between 51 and 60 yr. In symptomatic patients, there was a high degree of correlation between severity of H. ***pylori*** infection on histopathological examination and IgG ($P < 0.02$) levels. The use of IgG and IgA estimation could have identified H. ***pylori*** infection without endoscopy in 50 of the 83 patients. Serology for IgG and IgA ***antibodies*** against H. ***pylori*** may play a major role in decreasing the need for endoscopy, but cut-off values must be determined for each assay based on the prevalence of ***antibodies*** in the population.

L11 ANSWER 62 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 1999383622 EMBASE
TI Monitoring of Helicobacter ***pylori*** eradication by anti-H. ***pylori*** determination.
AU Antoljak N.; Vulkadinovic M.V.; Zubcic A.; Topic E.
CS Dr. N. Antoljak, Clinical Institute of Chemistry, Sestre Milosrdnice Univ. Hospital, Vinogradska c. 29, HR-10000 Zagreb, Croatia
SO Acta Clinica Croatica, (1999) 38/3 (203-207).
Refs: 10
ISSN: 0353-9466 CODEN: ACLCED
CY Croatia
DT Journal; Article
FS 004 Microbiology
005 General Pathology and Pathological Anatomy
027 Biophysics, Bioengineering and Medical Instrumentation
048 Gastroenterology
LA English
SL English; Serbo-Croatian
AB Helicobacter ***pylori*** (H. ***pylori***) infection results in ***gastric*** mucosa inflammation called chronic superficial ***gastritis*** , developing peptic ulceration in some patients. The two major categories of diagnostic tests are non-invasive tests and invasive methods using endoscopy. The aim of the study was to monitor the efficacy of anti-H. ***pylori*** by the non-invasive quantitative serologic method. The ELISA Pyloriset EIA-G (Orion Diagnostica, Finland) with a cut-off titer of 300 mg/L was used to measure the concentration of specific anti-H. ***pylori*** IgG in sera of 34 patients with positive dyspeptic illness history before, and then two, four and six months after the treatment. A titer decline by .gtoreq.40% of the ValUe measured before therapy is named seroconversion. The mean percentage of titer decline was greatest two months after the treatment (49%; $p<0.001$). A statistically significant decrease persisted after 4 and 6 months (66% and 78% of the pretherapeutic titer, respectively; $p<0.005$). After 6 month monitoring, 94% of patients were found to be successfully seroconverted. According to

some authors, a 4-month titer monitoring is needed to ascertain H. ***pylori*** eradication and to confirm seroconversion. Unlike these studies, our results showed that 73.4% of patients had a significant titer decline after 4 months, while 94% of patients were seroconverted after 6 months. So, the ***antibody*** titer decrease recorded 6 months following antimicrobial treatment could be an indicator of successful eradication in almost all treated patients.

L11 ANSWER 63 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 1999328131 EMBASE
TI Helicobacter ***antibodies*** in Finnish centenarians.
AU Rehnberg-Laiho L.; Louhija J.; Rautelin H.; Jusufovic J.; Tilvis R.; Miettinen A.; Kosunen T.U.
CS Dr. T.U. Kosunen, Haartman Institute, Dept. of Bacteriology and Immunology, University of Helsinki, Haartmaninkatu 3, 00014 Helsinki, Finland. timo.kosunen@helsinki.fi
SO Journals of Gerontology - Series A Biological Sciences and Medical Sciences, (1999) 54/8 (M400-M403).
Refs: 25
ISSN: 1079-5006 CODEN: JGASFW
CY United States
DT Journal; Article
FS 020 Gerontology and Geriatrics
048 Gastroenterology
LA English
SL English
AB Background. The prevalence of helicobacter ***antibodies*** increases with age and, in many developed countries, is highest in people born before 1940. Data on very old subjects are, however, limited. In this study we wanted to determine whether the age-related increase in the seroprevalence of H. ***pylori*** infection continues even in the oldest age group alive in Finland, the centenarians. Methods. Sera from 173 subjects (93% of all centenarians alive in Finland in 1991) were available for the present study. IgG and IgA ***antibodies*** against H. ***pylori*** were determined by an in-house enzyme ***immunoassay*** . To estimate the influence of atrophic ***gastritis*** on the prevalence of helicobacter ***antibodies*** , serum ***pepsinogen*** I (PG I) concentrations and parietal cell ***antibodies*** (PCAs) were measured by an enzyme ***immunoassay*** and indirect immunofluorescence, respectively. Results. The prevalence of helicobacter ***antibodies*** in Finnish centenarians was 66%. Low PG I values (<28 .mu.g/l) were found in 36% and positive PCAs in 16% of the subjects studied. The prevalence of PCAs was especially high (50%) in H. ***pylori*** -negative subjects with low PG I values, suggesting severe ***gastric*** atrophy. Conclusions. The age-related increase in H. ***pylori*** seroprevalence did not continue in the oldest age group alive in Finland. This may be explained partly by a relatively high frequency of atrophic ***gastritis*** (as suggested by low PG I values) in H. ***pylori*** -negative centenarians, but other factors - such as selective H. ***pylori*** -related mortality - may also have contributed to the fairly low seroprevalence (66%) observed.

L11 ANSWER 64 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 1999061826 EMBASE
TI Rapid immunochromatographic assay for detection of Helicobacter

pylori ***antibodies*** .

AU Pavlitou K.; Pastore F.; Moldovanidou K.; Gioula G.; Routsinas Ch.; Polidorou F.; Malaka E.

CS K. Pavlitou, Microbiol. 'Agios Demetrios' Dept., General Hospital, Thessaloniki, Greece

SO Acta Microbiologica Hellenica, (1998) 43/3 (267-270).
Refs: 12
ISSN: 0438-9573 CODEN: AMBHAA

CY Greece

DT Journal; Conference Article

FS 004 Microbiology
026 Immunology, Serology and Transplantation
048 Gastroenterology

LA Greek

SL English; Greek

AB The aim of the study was to evaluate the accuracy of a rapid immunochromatographic assay Helicobacter ***pylori*** EASY-card in the qualitative determination of Helicobacter ***pylori*** (HP) ***antibodies*** . A total of 311 patients undergoing endoscopy was studied. Diagnosis of lip infection was established if rapid urease test and smear's stain (haematoxylineosin, Giemsa) were positive. Anti-HP ***antibodies*** were detected in the serum of patients using Helicobacter ***pylori*** EASY-card and enzyme ***immunoassay*** (ELISA). The sensitivity and specificity of EASY-card and ELISA tests were 64%, 69% and 89%, 79% respectively. The above results indicate that Helicobacter ***pylori*** EASy-card is a rapid and useful test; however, it should be used only as preliminary, alternative, non-invasive procedure in patients with suspected HP infection.

L11 ANSWER 65 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

AN 1998419516 EMBASE

TI Positive association between Helicobacter ***pylori*** infection and food allergy in children.

AU Corrado G.; Luzzi I.; Lucarelli S.; Frediani T.; Pacchiarotti C.; Cavaliere M.; Rea P.; Cardi E.

CS Dr. G. Corrado, Paediatric Gastroenterology Unit, Paediatric Clinic Institute, La Sapienza University, Viale Regina Elena 324, I-00161 Rome, Italy

SO Scandinavian Journal of Gastroenterology, (1998) 33/11 (1135-1139).
Refs: 48
ISSN: 0036-5521 CODEN: SJGRA4

CY Norway

DT Journal; Article

FS 004 Microbiology
007 Pediatrics and Pediatric Surgery
026 Immunology, Serology and Transplantation
048 Gastroenterology

LA English

SL English

AB Background: In children Helicobacter ***pylori*** has been involved as a pathogenetic factor in ***gastritis*** and duodenal ulcer and as a cofactor in protein-losing enteropathy, chronic diarrhoea, short stature, and ***gastritis*** lymphoproliferative disease. A subset of an H. ***pylori*** strain possesses an antigen, CagA, as a virulence factor. In the present study we determined anti-H. ***pylori*** IgG and

anti-CagA IgG titres in children with food allergy. Methods: Ninety paediatric patients were studied: 30 with food allergy, 30 with atopic asthma, and 30 with inflammatory bowel disease. Anti-H. ***pylori*** IgG and anti-CagA IgG were determined in all children by means of a commercial enzyme ***immunoassay*** (ELISA). Results: The anti-H. ***pylori*** IgG titre was significantly higher in allergic patients than in the other two groups. The anti-CagA IgG titre did not differ significantly between the patients. Conclusions: These findings show a positive association between H. ***pylori*** infection and food allergy in children. We hypothesize that virulence factors other than CagA may be involved in the pathogenesis of H. ***pylori*** infection in paediatric patients with food allergy.

L11 ANSWER 66 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 1998383390 EMBASE
TI Comparison of salivary and serum enzyme ***immunoassays*** for the diagnosis of Helicobacter ***pylori*** infection.
AU Embil J.M.; Choudhri S.H.; Smart G.; Aldor T.; Pettigrew N.M.; Grahame G.R.; Dawood M.R.; Bernstein C.N.
CS C.N. Bernstein, Division of Gastroenterology, GB443 Health Sciences Center, 820 Sherbrok Street, Winnipeg, Man. R3A 1R9, Canada. cbernst@cc.umanitoba.ca
SO Canadian Journal of Infectious Diseases, (1998) 9/5 (277-280).
Refs: 15
ISSN: 1180-2332 CODEN: CJDIES
CY Canada
DT Journal; Article
FS 004 Microbiology
005 General Pathology and Pathological Anatomy
LA English
SL English; French
AB Infection with Helicobacter ***pylori*** has been established as an important risk factor for the development of peptic ulcer disease, ***gastritis*** and ***gastric*** caner. The diagnosis of H ***pylori*** infection can be established by invasive or noninvasive techniques. Two noninvasive enzyme ***immunoassays*** (EIAs) for ***antibody*** detection - HeliSal and ***Pylori*** State - were compared with histology. Both assays detect immunoglobulin (Ig) G directed against purified H ***pylori*** antigen. The test populations consisted of 104 consecutive patients scheduled for upper gastrointestinal endoscopy. Of these patients, 97 (93%) had symptoms compatible with peptic ulcer disease. Saliva and serum were collected simultaneously at the time of endoscopy. Salivary EIA had a sensitivity of 66%, specificity of 67%, positive predictive value of 67% and negative predictive value of 66% compared with the serum EIA, where the results were 98%, 48%, 64% and 96%, respectively. Although the salivary EIA is an appealing noninvasive test, it was not a sensitive and specific assay. The serum EIA also lacked specificity, but was highly sensitive with a good negative predictive value. Although a negative serum EIA rules out H ***pylori*** infection, a positive must be interpreted in the clinical context and confirmed with a more specific measure.

L11 ANSWER 67 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 1998057224 EMBASE
TI Comparison of rapid office-based serology with formal laboratory-based

ELISA testing for diagnosis of Helicobacter ***pylori*** ***gastritis***

AU Kroser J.A.; Faigel D.O.; Furth E.E.; Metz D.C.

CS Dr. D.C. Metz, Gastroenterology Division, 3 Ravdin Building, Univ. of Pennsylvania Medical Center, 3400 Spruce Street, Philadelphia, PA 19104, United States

SO Digestive Diseases and Sciences, (1998) 43/1 (103-108).
Refs: 37
ISSN: 0163-2116 CODEN: DDSCDJ

CY United States

DT Journal; Article

FS 004 Microbiology
029 Clinical Biochemistry
048 Gastroenterology

LA English

SL English

AB Accurate and cost-effective diagnosis of Helicobacter ***pylori*** ***gastritis*** has taken on major importance. Several serologic tests for the diagnosis of H. ***pylori*** infection are commercially available. We compared the performance of the FlexSure HP rapid IgG ***antibody*** test with the conventional HM-CAP ELISA to evaluate whether qualitative office-based serology is reliable enough to replace formal laboratory-based testing. We assessed H. ***pylori*** status by concordance in 100 consecutive patients with antral biopsy, rapid urease, and 1 .mu.Ci [14C]urea breath tests. Both ***antibody*** tests had good sensitivity and specificity (> 86%). Concordance between the two ***antibody*** tests occurred in 87/93 patients (94%). Based on our data, the office-based FlexSure HP performed equally well as the laboratory-based formal ELISA and may be a better choice for initial serologic diagnosis in untreated patients.

L11 ANSWER 68 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

AN 97327436 EMBASE

DN 1997327436

TI Relationship of helicobacter ***pylori*** infection to several malignant and non-malignant gastrointestinal diseases.

AU Erkisi M.; Colakoglu S.; Koksal F.; Tuncer I.; Burgut R.; Karakose H.; Doran F.; Zorludemir S.

CS M. Erkisi, Gazipasa Bulvari, Talay Apt. Kat:1, Adana, Turkey

SO Journal of Experimental and Clinical Cancer Research, (1997) 16/3 (289-294).
Refs: 21
ISSN: 0392-9078 CODEN: JECRDN

CY Italy

DT Journal; Article

FS 004 Microbiology
016 Cancer
017 Public Health, Social Medicine and Epidemiology
048 Gastroenterology

LA English

SL English

AB The importance of the Helicobacter ***Pylori*** infection was investigated as a risk factor for several gastrointestinal diseases. In this study 203 patients with ***gastric*** cancer, 61 with peptic ulcus, 60 with ***gastritis*** and 100 asymptomatic control subjects

were investigated. Serum samples were examined for IgC ***antibodies*** to H. ***pylori*** by enzyme linked ***immunoassay*** - tissue samples were stained for H. ***pylori*** by Wartin-Stary technique and by Giemsa for routine histopathology. H. ***pylori*** seropositivity was 58.1% in ***gastric*** cancer, 54% in peptic ulcus, 63.3% in ***gastritis*** and 27% in asymptomatic control group. There was a 10.1% discordance between the serum and tumor determinants in the seropositive group and 11.3% of discordance in the seronegative group. The frequency of H. ***pylori*** seropositivity was lowest in cardia tumors (22.7%) and highest in antral tumors (65.5%, p = 0.00002). H. ***pylori*** seropositivity was 29% in diffuse type of histology, 35% in mixed type and 79% in the intestinal type (p = 0.00000). In the ***gastric*** cancer patients the frequent use of salty food (p = 0.00001, OR = 6.4), excessive salt, pickled food (p = 0.0000, OR = 24.92) and proteins (p = 0.003, OR = 0.45) were more significant than asymptomatic volunteers. In ***gastric*** cancer patients the frequent use of salty and pickled food were relevantly associated with H. ***pylori*** infection (p = 0.001). It was concluded that H. ***pylori*** infection could play a role in the pathogenesis of non-malignant gastrointestinal diseases which may be the precursor of carcinoma. However, other contributing factors to carcinogenesis must be investigated.

L11 ANSWER 69 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 97325229 EMBASE
DN 1997325229
TI Immunoglobulin A ***antibodies*** to Helicobacter ***pylori*** .
AU Jaskowski T.D.; Martins T.B.; Hill H.R.; Litwin C.M.
CS T.D. Jaskowski, ARUPICEP, 500 Chipeta Way, Salt Lake City, UT 84108, United States
SO Journal of Clinical Microbiology, (1997) 35/11 (2999-3000).
Refs: 16
ISSN: 0095-1137 CODEN: JCMIDW
CY United States
DT Journal; Article
FS 004 Microbiology
026 Immunology, Serology and Transplantation
LA English
SL English
AB Serological testing for immunoglobulin G (IgG) ***antibodies*** to Helicobacter ***pylori*** has proven useful in supporting the diagnosis of infection with this organism, but the clinical value of IgA ***antibodies*** in H. ***pylori*** -related ***gastritis*** remains controversial. The purpose of our study was to determine the frequency of IgA-positive IgG-negative patients with symptoms of gastrointestinal (GI) disorders, thus assessing the clinical utility of IgA testing for H. ***pylori*** -related ***gastritis*** . It was found previously that the frequency of infected individuals in this category (IgA positive and IgG negative) is about 2%, but a large number of IgG-negative patients with GI disorders suggestive of H. ***pylori*** infection have not been investigated until now.

L11 ANSWER 70 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 97258518 EMBASE
DN 1997258518
TI Evaluation of salivary ***antibodies*** to detect infection with

Helicobacter ***pylori*** .
AU Loeb M.B.; Riddell R.H.; James C.; Hunt R.; Smaill F.M.
CS Dr. F.M. Smaill, University Medical Centre, 1200 Main Street West, Hamilton, Ont. L8N 3Z5, Canada. smaill@mcmaster.ca
SO Canadian Journal of Gastroenterology, (1997) 11/5 (437-440).
Refs: 22
ISSN: 0835-7900 CODEN: CJGAEJ
CY Canada
DT Journal; Article
FS 004 Microbiology
005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
048 Gastroenterology
LA English
SL English; French
AB Helicobacter ***pylori*** infection is an important cause of peptic ulcer disease and chronic ***gastritis*** . Infection with this bacterium stimulates the production of immunoglobulin (Ig) G ***antibody*** . Salivary IgG ***antibody*** tests to detect H ***pylori*** infection offer a convenient and noninvasive method of diagnosis. To evaluate an IgG salivary ***antibody*** kit, saliva was collected from 157 out-patients with dyspepsia referred for endoscopy to a tertiary centre. A salivary IgG ELISA ***antibody*** assay was performed using the Helisal Helicobacter ***pylori*** (IgG) assay kit, and at least four ***gastric*** biopsies were obtained. H ***pylori*** infection was confirmed by demonstration of the organism on Warthin-Starry silver stain (sensitivity 85%, specificity 55%). The prevalence of infection with H ***pylori*** was 30%. When the analysis was redone, excluding those treated with eradication therapy, the results were similar (sensitvity 86%, specificity 58%). The positive predictive value of the assay was 45% and the negative predictive value was 90%. Despite the ease of sampling, the assay used has limited diagnostic utility, lacking the predictive value to indicate which patients referred with dyspeptic symptoms to a tertiary care setting are infected with H ***pylori*** .

L11 ANSWER 71 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 96371022 EMBASE
DN 1996371022
TI Evaluation of whole blood ***antibody*** kit to detect active Helicobacter ***pylori*** infection.
AU Borody T.J.; Andrews P.; Shortis N.P.
CS Centre for Digestive Diseases, 144 Great North Road,Five Dock, NSW 2046, Australia
SO American Journal of Gastroenterology, (1996) 91/12 (2509-2512).
ISSN: 0002-9270 CODEN: AJGAAR
CY United States
DT Journal; Article
FS 004 Microbiology
005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
037 Drug Literature Index
048 Gastroenterology
LA English

SL English
AB Objectives: To evaluate the sensitivity and specificity of a whole blood ***antibody*** test (Helisal(TM) Rapid Blood test) for the detection of Helicobacter ***pylori*** using endoscopic diagnostic criteria of histology and urease tests as the 'gold standard.' Methods: A prospective trial of Helisal(TM) Rapid Blood (HRB) test was carried out in patients undergoing investigations for dyspepsia that included endoscopic biopsy for rapid urease test, microbiological culture, and histology. Blood samples were obtained at the time of endoscopy and were tested for the presence of ***antibody*** to H. ***pylori*** using the HRB test. In a separate patient group, results of ***antibody*** tests in whole venous and capillary blood were compared (n = 25). Results: The rapid blood test was carried out immediately after the endoscopic examination with a result available in under 10 min in all cases. In 203 patients examined, the HRB test detected 70 of 203 to be H. ***pylori*** positive as compared with 71 of 203 using urease/histology. Against combined urease/histology tests, the HRB test achieved 82% sensitivity and 91% specificity. Five patients were judged to be 'false negative' on endoscopic tests for H. ***pylori*** (extensive intestinal metaplasia n = 3; recent use of antimicrobials) yet the HRB test diagnosed the presence of infection, which could be shown to resolve on treatment. The HRB achieved 89% sensitivity and 91% specificity upon correctly including these five patients in the calculations. In all 25 patients tested, venous and capillary blood results concurred giving HRB test positivity in each case. Conclusions: Whether using whole venous or capillary blood, the HRB test is a quick, convenient, and accurate test for the diagnosis of active H. ***pylori*** infection in patients previously not treated. In a subgroup of patients with low level infection due to recent antimicrobials or intestinal metaplasia negative to all endoscopic tests, the blood test can still correctly diagnose H. ***pylori*** infection. Because blood samples require no centrifugation before testing, the greatest usefulness of this test will be that of a primary office diagnostic device.

L11 ANSWER 72 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 96150640 EMBASE
DN 1996150640
TI Comparison of rapid serological tests (FlexSure HP and QuickVue) with conventional ELISA for detection of Helicobacter ***pylori*** infection.
AU Graham D.Y.; Evans Jr. D.J.; Peacock J.; Baker J.T.; Schrier W.H.
CS Veterans Affairs Medical Center, 2002 Holcombe Blvd.,Houston, TX 77030, United States
SO American Journal of Gastroenterology, (1996) 91/5 (942-948). ISSN: 0002-9270 CODEN: AJGAAR
CY United States
DT Journal; Article
FS 004 Microbiology
006 Internal Medicine
048 Gastroenterology
LA English
SL English
AB Background: There is a need for accurate and rapid tests for Helicobacter ***pylori*** infection especially since the recent National Institutes of Health Consensus Development Conference on H. ***pylori*** in peptic ulcer disease charged the medical community with treating H.

pylori infection in all patients with H. ***pylori*** and ulcer disease. Methods: We prospectively compared a simple, rapid serological test (FlexSure HP, SmithKline Diagnostics) for the detection of serum IgG ***antibodies*** against H. ***pylori*** with another rapid test (QuickVue, Quidel) and two enzyme ***immunoassays*** (HM-CAP, Enteric Products, and PyloriStat, BioWhittaker). Serum samples from 551 individuals including both symptomatic patients (196) and asymptomatic volunteers (355) were tested for the presence of IgG ***antibodies*** against H. ***pylori*** . The presence or absence of active H. ***pylori*** infections was determined using the [14C]-urea breath test. Results: All of the serological tests performed well. FlexSure HP had calculated sensitivity, specificity, and accuracy of 94.4, 87.6, and 91.1%, respectively, relative to the urea breath test. In 49 of the 551 samples, the urea breath test and FlexSure HP did not agree. Those samples were tested with HM-CAP ***immunoassay*** to confirm presence or absence of IgG ***antibodies*** against H. ***pylori*** . After the resolution of the discordant results, the sensitivity, specificity, and accuracy of FlexSure HP were 96.0, 95.1, and 95.6%, respectively, and were comparable to HM-CAP and PyloriStat. FlexSure HP was compared with histology or culture in 75 cases, and the accuracy was 100%. FlexSure HP and QuickVue were compared using 200 serum samples. FlexSure HP was more specific (88.7 vs 79.4%) and accurate (91 vs 84%) than QuickVue ($p < 0.05$ for both), relative to the urea breath test with discordant samples unresolved: FlexSure HP was also simpler to use, easier to interpret, and faster than QuickVue. FlexSure HP required no sample dilution, one reagent, four steps, and 5 min to complete. Conclusion: FlexSure HP is an excellent option for in-office tests for the physician who desires immediate results or for small laboratories that do not have the volume of H. ***pylori*** testing to justify ELISA test formats.

L11 ANSWER 73 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 96053180 EMBASE
DN 1996053180
TI Helicobacter ***pylori*** and recurrent abdominal pain in children.
AU Hardikar W.; Feekery C.; Smith A.; Oberklaid F.; Grimwood K.
CS Department of Pediatrics, Yale University School of Medicine, 333 Cedar Street,New Haven, CT 06510, United States
SO Journal of Pediatric Gastroenterology and Nutrition, (1996) 22/2 (148-152).
ISSN: 0277-2116 CODEN: JPGND6
CY United States
DT Journal; Article
FS 007 Pediatrics and Pediatric Surgery
048 Gastroenterology
LA English
SL English
AB Recurrent abdominal pain is one of the most common presentations to pediatricians; yet an organic etiology can be found in only 10% of cases. Because infection with Helicobacter ***pylori*** in adults and children results in ***gastritis*** , a causative role for the organism has been postulated. To investigate this theory, we conducted a prospective case-control study in children with recurrent abdominal pain using serum H. ***pylori*** IgG ***antibodies*** measured by an enzyme immunoabsorbent assay. Age, sex, ethnicity, and socioeconomic status were adjusted in the statistical model. Five subjects (5.1%) and 14

controls (14.3%) had raised serum IgG ***antibodies*** to H. ***pylori*** (adjusted OR, 0.21; 95% confidence interval, 0.05, 0.85). The negative association between H. ***pylori*** and recurrent abdominal pain indicates that this organism is unlikely to have an important etiologic role in this disorder.

L11 ANSWER 74 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 95318303 EMBASE
DN 1995318303
TI A practical single sample dry latex agglutination test for Helicobacter ***pylori*** ***antibody*** detection.
AU Midolo P.D.; Lambert J.R.; Russell E.G.; Lin S.K.
CS Department of Microbiology, Monash Medical Centre, 246 Clayton Road,Clayton 3168, Vic., Australia
SO Journal of Clinical Pathology, (1995) 48/10 (969-971).
ISSN: 0021-9746 CODEN: JCPAAK
CY United Kingdom
DT Journal; Article
FS 004 Microbiology
005 General Pathology and Pathological Anatomy
048 Gastroenterology
LA English
SL English
AB Assessment of a single serum sample for Helicobacter ***pylori*** ***antibodies*** is frequently requested in routine diagnostic laboratories. Current enzyme linked immunosorbent assay (ELISA) kits are not ideal for testing small numbers of serum samples and some have low sensitivities, specificities or large grey zones.-A panel of 90 serum samples from patients who had presented for routine upper endoscopy was used to compare three kits for the detection of H ***pylori*** ***antibodies*** : (1) Pyloriset Dry, total ***antibody*** latex agglutination, Orion Diagnostica, Espoo, Finland; (2) Pyloriset enzyme ***immunoassay*** (EIA), IgG ELISA, Orion; and (3) Hel-p, IgG ELISA, Amrad, Kew, Victoria, Australia. Diagnosis of H ***pylori*** positivity was made if culture results and either rapid urease test or histopathology were positive. The sensitivity, specificity, positive, and negative predictive value for each test was as follows: Orion: latex 93.3%, 95.6%, 95.5%, 93.3%, respectively; Orion: EIA-G 84.4%, 97.8%, 97.4%, 84.4%, respectively; and Amrad: EIA-G 100%, 88.9%, 90%, 100%, respectively. The latex test performed better than the EIAs with respect to sensitivity and specificity.

L11 ANSWER 75 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 93326585 EMBASE
DN 1993326585
TI Prevalence of immunoglobulin G ***antibodies*** to Helicobacter ***pylori*** in Chilean individuals [7].
AU Figueroa G.; Troncoso M.; Portell D.P.; Toledo M.S.; Acuna R.; Arellano L.
CS Microbiology Unit, Inst Nutrition and Food Technology, University of Chile, Casilla 138-11,Santiago, Chile
SO European Journal of Clinical Microbiology and Infectious Diseases, (1993) 12/10 (795-797).
ISSN: 0934-9723 CODEN: EJCDEU
CY Germany
DT Journal; Letter

FS 004 Microbiology
017 Public Health, Social Medicine and Epidemiology
LA English

L11 ANSWER 76 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 93258309 EMBASE
DN 1993258309
TI Use of serum specific immunoglobulin ***antibodies*** to determine helicobacter ***pylori*** associated ***gastritis*** .
AU Fischbach W.; Wosnik K.; Kirchner T.; Mossner J.
CS Medizinische Poliklinik, University of Wurzburg, Klinikstr. 8,D-97070 Wurzburg, Germany
SO Zeitschrift fur Gastroenterologie, (1993) 31/7-8 (429-431).
ISSN: 0044-2771 CODEN: ZGASAX
CY Germany
DT Journal; Article
FS 004 Microbiology
020 Gerontology and Geriatrics
026 Immunology, Serology and Transplantation
048 Gastroenterology
LA English
SL English; German
AB A prospective study in 169 consecutive patients referred for upper gastrointestinal endoscopy was initiated to investigate the diagnostic performance of serum Helicobacter ***pylori*** (HP) specific immunoglobulin (Ig) G ***antibodies*** . Using an enzyme linked immunosorbent assay (ELISA) an excellent correlation between serologic evidence of HP and the demonstration of this organism by histology and urease test in 79 H P-positive patients was found. Serum IgG also correlated with the histological degree and the activity of ***gastritis*** . Our results demonstrate that serum IG G ***antibodies*** , as determined by ELISA, are highly useful for diagnosis of HP-associated ***gastritis*** .

L11 ANSWER 77 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 91315671 EMBASE
DN 1991315671
TI High prevalence of Helicobacter ***pylori*** infection and histologic ***gastritis*** in asymptomatic hispanics.
AU Dehesa M.; Dooley C.P.; Cohen H.; Fitzgibbons P.L.; Perez-Perez G.I.; Blaser M.J.
CS Depts. Medicine and Pathology, Univ. of Southern California, School of Medicine,Los Angeles, CA 90033, United States
SO Journal of Clinical Microbiology, (1991) 29/6 (1128-1131).
ISSN: 0095-1137 CODEN: JCMIDW
CY United States
DT Journal; Article
FS 004 Microbiology
005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
048 Gastroenterology
LA English
SL English
AB In this study, we estimated the prevalence of Helicobacter ***pylori*** infection and histologic ***gastritis*** in 58 asymptomatic Hispanic

adult volunteers (mean age, 41 years; 59% male) by endoscopic biopsy of the upper gastrointestinal tract. Forty-six subjects (79%) were found to harbor H. ***pylori*** in ***gastric*** biopsies, and all had histologic ***gastritis*** . Four other subjects were found to have ***gastritis*** in the absence of H. ***pylori*** . Similar prevalences of H. ***pylori*** and ***gastritis*** were noted in all age groups and also in American-born and immigrant Hispanics. Biopsy data and serologic studies of H. ***pylori*** ***antibodies*** correlated well. We conclude that H. ***pylori*** infection is an almost universal finding in the ***gastric*** mucosa of asymptomatic adult Hispanics, regardless of age. The clinical significance of these findings is unknown, but we speculate that H. ***pylori*** and its associated ***gastritis*** could have a role in the high incidence of ***gastric*** carcinoma in Hispanic populations.

L11 ANSWER 78 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 91312270 EMBASE
DN 1991312270
TI A solid-phase enzyme-linked immunospot (ELISPOT) assay for detection of Helicobacter ***pylori*** ***antibody*** -producing cells in ***gastric*** mucosa.
AU Sugiyama T.; Furuyama S.; Awakawa T.; Imai K.; Yabana T.; Yachi A.; Yokota Oguma K.K.
CS Dept. of Internal Medicine, Sapporo Medical College, S-l, W-16, Chuo-ku,Sapporo 060, Japan
SO Gastroenterologia Japonica, (1991) 26/5 (684).
ISSN: 0435-1339 CODEN: GAJABC
CY Japan
DT Journal; Article
FS 004 Microbiology
026 Immunology, Serology and Transplantation
048 Gastroenterology
LA English

L11 ANSWER 79 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 90034384 EMBASE
DN 1990034384
TI Value of serology (ELISA and immunoblotting) for the diagnosis of Campylobacter ***pylori*** infection.
AU Pena A.S.; Endtz Ph. H.; Offerhaus G.J.A.; Hoogenboom-Verdegaal A.; Van Duijn W.; De Vargas N.; Den Hartog G.; Kreuning J.; Van der Reyden J.; Mouton R.P.; Lamers C.B.H.W.
CS Department of Gastroenterology, Leiden University Hospital, P.O. Box 9600,NL-2300 RC Leiden, Netherlands
SO Digestion, (1989) 44/3 (131-141).
ISSN: 0012-2823 CODEN: DIGEBW
CY Switzerland
DT Journal; Article
FS 029 Clinical Biochemistry
048 Gastroenterology
LA English
SL English
AB Fifty-two unselected patients referred to for upper gastrointestinal endoscopy were evaluated in several ways to determine the presence of Campylobacter ***pylori*** . ***Antibodies*** against this

microorganism were measured to assess the value of serology for the diagnosis of C. ***pylori*** infection. Five antral biopsy specimens were taken in each patient for culture and bacteriological determinations, histology [morphology and Warthin-Starry (WS) staining] and the urease test (2, 3 and 24 h). Serum ***antibodies*** against a sonicate of 6 strains of microorganisms were assayed by enzyme-linked ***immunoassay*** (ELISA) and an immunoblotting technique. In 14 of the 52 patients the histology of the antrum was normal, 18 patients had chronic active ***gastritis*** and 20 had chronic ***gastritis*** without polymorphonuclear infiltration. In the group with normal histology, only 1 patient was positive for C. pilory with all methods, and 1 other subject was positive for IgG and 2 for IgA only with ELISA. In the group with chronic active ***gastritis*** , 14 were positive with all methods, 1 was negative by WS only and another was negative for IgA according to ELISA, WS and ***antibodies*** . Among the patients with chronic ***gastritis*** , 7 were positive and 7 negative with all tests; in the other 6 patients the results obtained with the various tests were divergent. Four serological tests were studied and validated against culture, WS and urease test which were considered to be the reference methods. The serological tests showed high sensitivity and specificity for the detection of C. ***pylori*** -associated active chronic ***gastritis*** of the antrum, and can therefore serve as noninvasive methods to identify individuals with this condition.

L11 ANSWER 80 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 89163233 EMBASE
DN 1989163233
TI Serum IgG and IgA ***antibody*** responses to campylobacter ***pylori*** in a group of healthy asymptomatic volunteers.
AU Westblom T.U.; Barthel J.S.; Kosunen T.U.; Everett E.D.
CS Department of Medicine, Section of Infectious Diseases, Marshall University School of Medicine, Huntington, WV 25755-9410, United States
SO Scandinavian Journal of Infectious Diseases, (1989) 21/3 (311-314). ISSN: 0036-5548 CODEN: SJIDB7
CY Sweden
DT Journal
FS 004 Microbiology
026 Immunology, Serology and Transplantation
048 Gastroenterology
LA English
SL English
AB Sera from 17 healthy asymptomatic volunteers were tested for presence of IgG and IgA ***antibodies*** against Campylobacter ***pylori*** and correlated with endoscopic biopsy findings. Three volunteers infected with C. ***pylori*** had the highest IgG ***antibody*** titers of the group. None of 14 C. ***pylori*** free subjects had significant IgG ***antibody*** levels. IgA ***antibody*** titers were negative in all subjects regardless of state of infection, in contrast to control sera from symptomatic C. ***pylori*** infected patients who manifested high IgA ***antibody*** levels.

L11 ANSWER 81 OF 184 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 89071885 EMBASE
DN 1989071885
TI Age-dependent increase of Campylobacter ***pylori***

antibodies in blood donors.
AU Kosunen T.U.; Hook J.; Rautelin H.I.; Myllyla G.
CS Department of Bacteriology and Immunology, University of Helsinki, 00290 Helsinki, Finland
SO Scandinavian Journal of Gastroenterology, (1989) 24/1 (110-114). ISSN: 0036-5521 CODEN: SJGRA4
CY Norway
DT Journal
FS 048 Gastroenterology
LA English
SL English
AB ***Antibodies*** against Campylobacter ***pylori*** were determined in 500 blood donors aged 18 to 65 years. Acid extract from a C. ***pylori*** strain was used as antigen in enzyme ***immunoassay*** . The proportion of donors with high ***antibody*** titers increased with age. For IgG ***antibodies*** it was 10% in the age group from 18 to 25 years but 60% in the group from 56 to 65 years; the increase for IgA and IgM ***antibodies*** was from 5 to 42% and from 7 to 21%, respectively. The geometric mean titers of those with high values showed no clear changes with age, which would imply chronic antigenic stimulus.

L11 ANSWER 82 OF 184 LIFESCI COPYRIGHT 2001 CSA
AN 97:17620 LIFESCI
TI Campylobacter ***pylori*** antigens and uses thereof for detection of Campylobacter ***pylori*** infection
CS ENTERIC RESEARCH LABORATORIES, INC.
SO (1995) . US Patent 5459041; US Cl. 435/7.21 435/7.3 435/7.92 435/7.93 435/7.94 435/7.95 435/961 435/974 435/975 436/518 436/527 436/528 436/529 436/531 436/533 436/547 436/804 530/350 530/.
DT Patent
FS A; W3
LA English
AB Antigenic compositions are disclosed for use in diagnostic kits and the like for detecting the presence of ***antibodies*** specific for Campylobacter ***pylori*** , bacteria often associated with the occurrence of Type B ***gastritis*** and peptic ulcer disease. Samples of bodily fluids, for instance, may be contacted with immobilized antigen which is then washed and tested for the occurrence of significant levels of antigen/ ***antibody*** complex. Levels exceeding a predetermined positive threshold are indicative of ***antibodies*** to Campylobacter ***pylori*** in the sample tested. Kits employing the antigenic compositions of the invention preferably include means for detecting the antigen/ ***antibody*** complex such as materials and reagents for conducting an enzyme-linked immunosorbent assay, Western blot technique, liposome-based assay or other known detection tests.

L11 ANSWER 83 OF 184 LIFESCI COPYRIGHT 2001 CSA
AN 89:107753 LIFESCI
TI Time-resolved fluoroimmunoassay for Campylobacter ***pylori*** ***antibodies*** .
AU Aceti, A.; Pennica, A.; Leri, O.; Caferro, M.; Grilli, A.; Celestino, D.; Casale, V.; Citarda, F.; Grassi, A.; Sciarretta, F.
CS Inst. Trop. and Infect. Dis., La Sapienza Univ., 00161 Rome, Italy
SO LANCET., (1989) vol. 2, no. 8661, p. 505.
DT Journal

FS J; F
LA English
AB Dr. Loffeld and colleagues suggest that an ELISA test for Campylobacter ***pylori*** ***antibodies*** might replace endoscopy in the diagnosis of ***gastritis*** associated with this bacterium. However, with a cut-off of optical density (OD) greater than 2 multiplied by 1 the ELISA had a specificity of 100% and a sensitivity of 85 multiplied by 4%; at a lower cut-off (OD above 1) the sensitivity was 100% but the specificity fell to 72 multiplied by 7%. We have evaluated a time-resolved fluoroimmunoassay (TR-FIA), to detect C. ***pylori*** ***antibodies*** . This test is based on the labelling of ***antibodies*** with europium (Eu) and conversion of the specifically bound non-fluorescent label to highly fluorescent chelate solution, followed by measurement with a time-resolved fluorimeter. TR-FIA was compared with ELISA.

L11 ANSWER 84 OF 184 MEDLINE
AN 2000451530 MEDLINE
DN 20460284
TI Accuracy of an enzyme ***immunoassay*** for the detection of Helicobacter ***pylori*** in stool specimens in the diagnosis of infection and posttreatment check-up.
AU Forne M; Dominguez J; Fernandez-Banares F; Lite J; Esteve M; Gali N; Espinos J C; Quintana S; Viver J M
CS Department of Gastroenterology, Hospital Universitari Mutua de Terrassa, Barcelona, Spain.
SO AMERICAN JOURNAL OF GASTROENTEROLOGY, (2000 Sep) 95 (9) 2200-5. Journal code: 3HE. ISSN: 0002-9270.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200012
EW 20001203
AB OBJECTIVE: The aim of this study was to assess the reliability of a newly developed enzyme ***immunoassay*** for Helicobacter ***pylori*** -specific antigen detection in stools (HpSA) compared to other standardized diagnostic techniques such as histology (H), rapid urease test (RUT) and 13C-urea breath test (UBT) to diagnose H. ***pylori*** infection and to evaluate its usefulness in determining H. ***pylori*** status after treatment. METHODS: One hundred eighty-eight patients referred to our department for upper gastrointestinal endoscopy were included. H. ***pylori*** infection was confirmed in all patients by HpSA test in stools, RUT, UBT, and H. Patients were defined as positive for H. ***pylori*** if RUT and UBT or H were positive. A total of 142 symptomatic patients received eradication treatment and were reassessed 6 wk after therapy; for 70 of these patients, stool samples were also collected at 24 h and 6 months after finishing eradication treatment. In the posttreatment follow-up, UBT was used as gold standard. RESULTS: The sensitivity of HpSA test for the diagnosis of H. ***pylori*** infection using a cut-off value of 0.130 was 89.5% and its specificity 77.8%. This specificity was lower than that obtained with UBT, H, and RUT. In the early follow-up the sensitivity of HpSA test was null. At 6 weeks and at 6 months post-treatment its sensitivity was 70.4% and 50% and its specificity was 81.6% and 79.3%, respectively. CONCLUSIONS: The HpSA stool

test, using a cut-off value of 0.130, may be useful for the primary diagnosis of H. ***pylori*** infection, with sensitivity similar to that obtained with other standard tests, but with less specificity. HpSA test is not useful for early monitoring of treatment efficacy. At 6 wk and at 6 months posttreatment, HpSA test lacks accuracy as compared to UBT for evaluating the outcome of the eradication treatment.

L11 ANSWER 85 OF 184 MEDLINE
AN 2000386919 MEDLINE
DN 20319644
TI Study of diagnostic modalities and pathology of Helicobacter ***pylori*** infection in children.
AU Bansal D; Patwari A K; Logani K B; Malhotra V L; Anand V K
CS Department of Pediatrics, Lady Hardinge Medical College, New Delhi.
SO INDIAN JOURNAL OF PATHOLOGY AND MICROBIOLOGY, (1999 Jul) 42 (3) 311-5. Journal code: GKK. ISSN: 0377-4929.
CY India
DT Journal; Article; (JOURNAL ARTICLE)
LA English
EM 200010
EW 20001002
AB To evaluate various diagnostic tests for Helicobacter ***pylori*** (Hp) in children, and to study the spectrum of endoscopic and histological changes in the stomach and duodenum of children with gastroduodenal disorders, associated with Hp infection Children below 12 years of age with various gastroduodenal disorders requiring upper gastrointestinal endoscopy were studied. Endoscopic biopsy specimens were collected from duodenum and antrum. Apart from histopathological examination of biopsy material, rapid urease test (RUT) of the antral biopsy specimen and blood examination to estimate specific IgG ***antibodies*** to Hp by Indirect Solid Phase Enzyme ***Immunoassay*** was performed. Forty seven children were included. Nine (19.1%) of them were positive both by serology and RUT. Seven (14.9%) were positive by histology. A significant correlation of Hp was noticed with chronic antral ***gastritis*** ($p = 0.002$) and chronic duodenitis ($p = 0.006$). Age equal to or more than 10 years was found to be significant risk factor for acquiring Hp infection. Prevalence of Hp in children with gastroduodenal complaints was found to be 19%. Both RUT and serology were found to be reliable diagnostic tests for Hp as compared with histology. Antral ***gastritis*** and chronic duodenitis had a significant correlation with Hp colonization.

L11 ANSWER 86 OF 184 MEDLINE
AN 2000282994 MEDLINE
DN 20282994
TI Plaunotol suppresses interleukin-8 secretion induced by Helicobacter ***pylori*** : therapeutic effect of plaunotol on H. ***pylori*** infection.
AU Takagi A; Koga Y; Aiba Y; Kabir A M; Watanabe S; Ohta-Tada U; Osaki T; Kamiya S; Miwa T
CS Department of Internal Medicine, Tokai University School of Medicine, Isehara, Kanagawa, Japan.. takagia@is-icc.u-tokai.ac.jp
SO JOURNAL OF GASTROENTEROLOGY AND HEPATOLOGY, (2000 Apr) 15 (4) 374-80. Journal code: A6J. ISSN: 0815-9319.
CY Australia
DT Journal; Article; (JOURNAL ARTICLE)

LA English
FS Priority Journals
EM 200009
EW 20000905
AB BACKGROUND: It has been suggested that ***gastric*** mucosal injury induced by Helicobacter ***pylori*** infection is mediated by interleukin-8 (IL-8). METHODS: We studied the effect of plaunotol, a drug extracted from the Plau-noi tree of Thailand, and reported it to be effective in the treatment of ulcers, of IL-8 secretion induced by H. ***pylori*** and of the inhibitory adhesion activity of the bacterium to ***gastric*** epithelial cells. Moreover, the therapeutic effect of plaunotol on H. ***pylori*** infection was assessed by using the gnotobiotic murine model. RESULTS: Plaunotol inhibited the growth of H. ***pylori*** (1.5 x 10(4) c.f.u./mL) at high doses (24-48 microg/mL), but not at low doses (3-6 microg/mL). Interleukin-8 secretion induced by H. ***pylori*** was inhibited by coculture with plaunotol in a dose-dependent manner. The adhesion of H. ***pylori*** to MKN45 cells was also suppressed by coculture with plaunotol in a dose-dependent manner. An in vivo study showed that plaunotol improved histological ***gastritis*** and decreased the H. ***pylori*** ***antibody*** titre. CONCLUSIONS: These findings suggest that plaunotol has a therapeutic effect on ***gastritis*** induced by H. ***pylori*** .

L11 ANSWER 87 OF 184 MEDLINE
AN 2000131489 MEDLINE
DN 20131489
TI Evaluation of an enzyme ***immunoassay*** for detecting Helicobacter ***pylori*** antigens in human stool samples.
AU Agha-Amiri K; Mainz D; Peitz U; Kahl S; Leodolter A; Malfertheiner P
CS Department of Gastroenterology, Hepatology and Infectious Diseases, Otto-von-Guericke-University, Magdeburg, Germany.
SO ZEITSCHRIFT FUR GASTROENTEROLOGIE, (1999 Dec) 37 (12) 1145-9. Journal code: XU1. ISSN: 0044-2771.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200005
EW 20000501
AB BACKGROUND AND AIM: So far, the detection of Helicobacter ***pylori*** (Hp) infection by stool analysis appeared to be almost impossible. With the Premier Platinum HpSA EIA a new enzyme ***immunoassay*** was developed for diagnosis of Hp infection, using polyclonal ***antibodies*** against Hp antigens in human stool. We evaluated this new test in its diagnostic accuracy in comparison to established reference methods. METHODS: From 54 consecutive patients (29 male, 25 female, age: 19 to 85 years) undergoing routine upper gastrointestinal endoscopy antral and corpus biopsies were taken for histology and Helicobacter urease test (HUT). Endoscopy, 13C-urea breath test (13C-UBT), serology, and stool probes sampling were performed within two days. Stool samples were aliquoted after reception and stored frozen (-20 degrees C) until tested. The Premier Platinum HpSA test (Meridian, Connecticut, Ohio, USA) was performed according to the manufactures protocol. Patients were considered to be infected with Hp if two of the four reference tests were positive. RESULTS: 28 of the 54 patients were Hp-infected. Only one of these was

found to be false-negative by the HpSA EIA. Two false-positive results were obtained in the noninfected group (sensitivity 96.4%, specificity 92.3%). CONCLUSION: In this group of patients investigated, the novel HpSA Enzyme ***Immunoassay*** (EIA) proved to be highly accurate for diagnosis of Hp infection. Collection and testing of stool are noninvasive and easy to perform, therefore this test will become an important tool for diagnosing Hp infection in clinical practice.

L11 ANSWER 88 OF 184 MEDLINE
AN 2000033670 MEDLINE
DN 20033670
TI Evaluation of three commercial serological tests with different methodologies to assess Helicobacter ***pylori*** infection.
AU van Der Ende A; van Der Hulst R W; Roorda P; Tytgat G N; Dankert J
CS Department of Medical Microbiology, Amsterdam, The Netherlands.
SO JOURNAL OF CLINICAL MICROBIOLOGY, (1999 Dec) 37 (12) 4150-2.
Journal code: HSH. ISSN: 0095-1137.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200003
EW 20000303
AB The sera of 142 Helicobacter ***pylori*** -positive and 32 H. ***pylori*** -negative patients were assessed by a desktop test (QuickVue), an enzyme-linked immunosorbent assay (ELISA) (HM-CAP), and a solid-phase, two-step chemiluminescent enzyme ***immunoassay*** (Immulite). These tests yielded sensitivities of 97, 97, and 91% and specificities of 97, 94, and 100%, respectively. In conclusion, the desktop test and the ELISA are more sensitive than the chemiluminescent enzyme ***immunoassay*** ($P < 0.05$). The chemiluminescent enzyme ***immunoassay*** has the advantage that it is fully automated.

L11 ANSWER 89 OF 184 MEDLINE
AN 1999405995 MEDLINE
DN 99405995
TI Helicobacter ***pylori*** ***antibody*** profile in household members of children with H. ***pylori*** infection.
AU Elitsur Y; Adkins L; Saeed D; Neace C
CS Department of Pediatrics, Marshall University, School of Medicine, Huntington, WV 25701-0195, USA.
SO JOURNAL OF CLINICAL GASTROENTEROLOGY, (1999 Sep) 29 (2) 178-82.
Journal code: IBG. ISSN: 0192-0790.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199912
EW 19991204
AB Intrafamilial spread is implicated as a major route for acquisition of Helicoobacter ***pylori*** infection. Investigating H. ***pylori*** cytotoxin-associated protein (CagA) and vacuolating toxin (VacA) ***antibodies*** within family members enabled the authors to evaluate this possibility further. Serum samples were collected prospectively from household members after their index children were diagnosed with active H.

pylori infection. Serum samples were evaluated for anti-H. ***pylori*** immunoglobulin G ***antibody*** using the enzyme ***immunoassay*** (IEA) method and for H. ***pylori*** CagA and VacA ***antibodies*** with the commercially available immunoprobing Western blot kit. Ten different families participated in the study, including 10 pediatric patients and 31 household members. All patients and 28 household members (90%) were seropositive for H. ***pylori*** ***antibody*** by IEA and Western blot tests. Overall, 17 subjects (41.4%) were CagA positive, 14 (34.1%) were VacA positive, 11 (26.8%) were positive for both ***antibodies*** , and 22 (53.6%) were negative for both ***antibodies*** . A significant association in bacterial ***antibody*** profile was found between the patient index members and all household members (Cohen's kappa and Mentel-Haenszel methods). In four families, more than 66% of the household members harbored the same ***antibody*** profile, and in two families a completely different profile was observed. Moreover, a similar H. ***pylori*** ***antibody*** profile between the index patient and the mother was found in six families, and between the index patient and the father in two families. The data strongly suggest an intrafamiliar transmission for H. ***pylori*** infection.

L11 ANSWER 90 OF 184 MEDLINE
AN 1999388378 MEDLINE
DN 99388378
TI New immunological assays for the diagnosis of Helicobacter ***pylori*** infection.
AU Vaira D; Holton J; Menegatti M; Ricci C; Landi F; Ali' A; Gatta L; Acciardi C; Farinelli S; Crosatti M; Berardi S; Miglioli M
CS Department of Internal Medicine, University of Bologna, Bologna, Italy.
SO GUT, (1999 Jul) 45 Suppl 1 I23-7. Ref: 51
Journal code: FVT. ISSN: 0017-5749.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199911
EW 19991105
AB There are several types of immunological tests available for the diagnosis and management of Helicobacter ***pylori*** infection. Most commercially available serological kits use the enzyme linked immunosorbent assay (ELISA) test format. Originally the kits used crude antigen preparations although many of the newer kits use a more purified antigen preparation, with often increased specificity but lower sensitivity. Near patient test kits are based either on latex agglutination or immunochromatography. Generally they have low sensitivities compared with laboratory tests. Western blotting, ELISA, and recombinant immunoblot assays (RIBA) have also been developed into commercially available kits and can be used to indicate the presence of specific virulence markers. An antigen detection kit has been developed for the detection of Helicobacter ***pylori*** in faeces. Immunological reagents have also been combined with other diagnostic modalities to develop immunohistochemical stains and DNA ***immunoassays*** . Helicobacter ***pylori*** is now recognised as

the cause of ***gastritis*** and most cases of peptic ulcer disease (PUD); its long term carriage increases the risk of ***gastric*** adenocarcinoma sixfold and it is designated as a class I carcinogen. H ***pylori*** has also been implicated as a cause of ***gastric*** mucosa associated lymphoid tissue lymphomas. Its relation to non-ulcer dyspepsia remains controversial. Additionally, long term carriage of the organism may be associated with short stature in young girls and, in the general population, as a possible risk factor for the development of vasospastic disorders and possibly skin immunopathology such as urticaria. With the recognition of H ***pylori*** as an important human pathogen, it has become one of the growing number of organisms to have its complete genome sequence mapped. Serology is an important method of determining colonisation status and can be used for diagnosis, as a screening procedure, or to follow the efficacy of eradication regimens. Most serological assays are in the ELISA format although some are based on the latex agglutination reaction. These latter are used principally as near patient assays. Most assays detect IgG in serum although some detect serum IgA. More recently developed assays detect IgA in saliva and the production of affinity purified ***antibodies*** has led to the development of an antigen detection assay for faecal specimens. Serological reagents have also been used in immunocytochemistry and to speed up the detection of amplified products of the polymerase chain reaction (PCR)-DNA ***immunoassays*** .

L11 ANSWER 91 OF 184 MEDLINE
AN 1999359814 MEDLINE
DN 99359814
TI Long-term follow-up study of serum immunoglobulin G and immunoglobulin A ***antibodies*** after Helicobacter ***pylori*** eradication.
AU Kato S; Furuyama N; Ozawa K; Ohnuma K; Iinuma K
CS Department of Pediatrics, Tohoku University School of Medicine, Japan.. skato@ped.med.tohoku.ac.jp
SO PEDIATRICS, (1999 Aug) 104 (2) e22.
Journal code: CZE. ISSN: 1098-4275.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199910
EW 19991003
AB OBJECTIVE: There have been few studies concerning serum titers of anti-Helicobacter ***pylori*** immunoglobulin G (IgG) ***antibody*** >12 months after eradication of the original infection. Moreover, clinical usefulness of immunoglobulin A (IgA) ***antibody*** levels remains to be established. The purpose of this study was to investigate long-term responses of serum IgG-specific and IgA-specific ***antibodies*** to H ***pylori*** in children after eradication therapy. STUDY DESIGN: A total of 34 children, 2 to 17 years of age (mean: 11.7 years) with H ***pylori*** -associated gastroduodenal disease received eradication therapy (proton pump inhibitor-based dual or triple regimens). Diagnoses included nodular ***gastritis*** (n = 8), ***gastric*** ulcer (n = 7), and duodenal ulcer (n = 19). Upper gastrointestinal endoscopy and biopsy were performed before the therapy and at 1 to 2 months' posttreatment. H ***pylori*** infection and eradication were defined by biopsy-based tests; eradication was successful in 28 patients and

unsuccessful in 6. Pretreatment IgG was positive in 30 patients (88. 2%), and the IgA was positive in 31 (91.2%), who were entered into this study (duration </=24 months). Serum samples were obtained before treatment and at 1, 3, 6, 12, 18, and 24 months' posttreatment. IgG and IgA ***antibodies*** were measured using commercial enzyme ***immunoassay*** kits (HM-CAP and PP-CAP; Enteric Products, Inc, New York, NY). RESULTS: Compared with pretreatment values, IgG and IgA ***antibodies*** significantly and steadily decreased at 1 through 24 months' posttreatment in successfully treated patients. A decrease in titer of the IgA class was significantly greater than that of the IgG class at 1 to 12 months' follow-up. There was no significant decrease in titer of either ***antibody*** in all but 2 patients with eradication failure. A >/=30% decrease in titer of the IgA ***antibody*** at 6 months indicated eradication with sensitivity of 90.5% and specificity of 100%. For the IgG ***antibody*** , a 30% decrease at 12 months showed equal sensitivity and specificity. Seroreversion rates of IgG and IgA ***antibodies*** were 53% and 48% at 12 months and were 86% and 81% at 24 months, respectively. The mean periods from the completion of eradication therapy to seroreversion of IgG and IgA ***antibodies*** were 11.2 +/- 7.0 and 11.6 +/- 7.8 months, respectively (not significantly different). A higher pretreatment titer of IgG ***antibody*** was related to a longer period of seroreversion (r = 0.44). In one patient, (13)C-urea breath test-confirmed reinfection was accompanied by reappearance of significant titers of the IgG and IgA ***antibodies*** . CONCLUSIONS: A serology test is useful for evaluating eradication in children. Approximately half of patients with successful eradication remained to be IgG-seropositive and IgA-seropositive at 12 months' posttreatment. When a decrease titer in ***antibody*** is used for assessing eradication, an endpoint of >/=6 months is required. The IgA ***antibody*** may be a more convenient indicator of H ***pylori*** status than is the IgG ***antibody*** .

L11 ANSWER 92 OF 184 MEDLINE
AN 1999311132 MEDLINE
DN 99311132
TI Prevalence of CagA, VacA ***antibodies*** in symptomatic and asymptomatic children with Helicobacter ***pylori*** infection.
AU Elitsur Y; Neace C; Werthammer M C; Triest W E
CS Department of Pediatrics, Marshall University School of Medicine, Huntington, West Virginia 25701-0195, USA.
SO HELICOBACTER, (1999 Jun) 4 (2) 100-5.
Journal code: CY4. ISSN: 1083-4389.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199910
EW 19991001
AB BACKGROUND: Limited data are available on the prevalence of CagA and VacA Helicobacter ***pylori*** ***antibodies*** in children. The aim of this study was to investigate the ***antibody*** prevalence to the H. ***pylori*** virulence factors CagA and VacA in symptomatic and asymptomatic children with H. ***pylori*** infection and to correlate these ***antibodies*** with the severity of ***gastric*** inflammation or density of H. ***pylori*** organisms in the

gastric mucosa. MATERIALS AND METHODS: Twenty-three symptomatic children and 132 asymptomatic children with positive H. ***pylori*** serology participated in this study. Anti-H. ***pylori*** IgG ***antibody*** and CagA or VacA H. ***pylori*** ***antibodies*** were measured by enzyme ***immunoassay*** (HM-CAP; sensitivity and specificity > 90%) and Western immunoblot (Helicoblot 2.0) methods, respectively. ***Gastric*** inflammation and H. ***pylori*** density were graded histologically using the revised Sydney criteria. RESULTS: The prevalence of CagA and VacA ***antibodies*** were 69% and 35% in symptomatic children and 54% and 52% in asymptomatic children, respectively. Multiple regression analysis showed a correlation between CagA ***antibody*** and the severity of ***gastritis*** but no correlation with other histological features, including the number of neutrophils or lymphoid follicles. Neither ***antibody*** correlated with the degree of bacterial density in the ***gastric*** mucosa. CONCLUSION: CagA and VacA H. ***pylori*** ***antibodies*** are common in the pediatric population. The combined CagA/VacA ***antibodies*** correlated weakly with the degree of mucosal inflammation.

L11 ANSWER 93 OF 184 MEDLINE
AN 1999129624 MEDLINE
DN 99129624
TI Helicobacter ***pylori*** infection in children with celiac disease: prevalence and clinicopathologic features.
AU Luzza F; Mancuso M; Imeneo M; Mesuraca L; Contaldo A; Giancotti L; La Vecchia A M; Docimo C; Pensabene L; Strisciuglio P; Pallone F; Guandalini S
CS Dipartimento di Medicina Sperimentale e Clinica, Universit`a di Catanzaro Magna Graecia, Italy.
SO JOURNAL OF PEDIATRIC GASTROENTEROLOGY AND NUTRITION, (1999 Feb) 28 (2) 143-6.
Journal code: JL6. ISSN: 0277-2116.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199906
EW 19990601
AB BACKGROUND: Celiac disease is frequently associated with chronic ***gastritis*** . Helicobacter ***pylori*** is the main etiologic agent of chronic ***gastritis*** . The aim of this study was to assess the prevalence of H. ***pylori*** , the related symptoms, and the endoscopic and histologic ***gastric*** features in children with celiac disease. METHODS: Eight-one (24 boys, 57 girls; age range: 1.4-17.7 years, median 6.8) children with celiac disease were studied. All children had a blood sample taken. In a subgroup of 30 children who underwent endoscopy, three ***gastric*** biopsy specimens were taken for histology (hematoxylin and eosin, Giemsa, immunohistochemistry) and urease quick test. Symptom complaints were recorded. Age- and sex-matched (one case, one control) children without celiac disease were used for comparison. Serum H. ***pylori*** IgG were measured by means of a locally validated commercial enzyme-linked ***immunoassay*** . RESULTS: Overall, 15 of 81 (18.5%) children with celiac disease and 14 of 81 (17.3%) control children were positive for H. ***pylori*** . The

percentage of H. ***pylori*** positivity was similar in children with untreated and treated celiac disease. Recurrent abdominal pain was the only symptom that helped to distinguish between H. ***pylori*** -positive and H. ***pylori*** -negative children. However, symptoms disappeared in patients with celiac disease after gluten withdrawal, irrespective of H. ***pylori*** status. All endoscopic (erythema, nodularity) and histologic (superficial-, interstitial-, lymphocytic- ***gastritis*** , activity, lymphoid follicles) findings did not differ between celiac and nonceliac H. ***pylori*** -positive children. CONCLUSIONS: Prevalence and clinical expressivity of H. ***pylori*** infection is not increased in children with celiac disease. The clinicopathologic pattern of the infection is not specifically influenced in this condition.

L11 ANSWER 94 OF 184 MEDLINE
AN 1998443826 MEDLINE
DN 98443826
TI Helicobacter ***pylori*** infection in recurrent abdominal pain.
AU Bansal D; Patwari A K; Malhotra V L; Malhotra V; Anand V K
CS Department of Pediatrics and Microbiology, Lady Hardinge Medical College, New Delhi.
SO INDIAN PEDIATRICS, (1998 Apr) 35 (4) 329-35.
Journal code: GM2. ISSN: 0019-6061.
CY India
DT (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LA English
EM 199901
EW 19990104
AB OBJECTIVE: To study the relationship between Helicobacter ***pylori*** (Hp) infection and recurrent abdominal pain (RAP) and to evaluate various modalities to diagnose Hp infection. DESIGN: Prospective case control study. SETTING: Teaching hospital. METHODS: Children between 3-12 years of age with RAP in whom upper gastrointestinal endoscopic examination was indicated were studied. Endoscopic biopsy specimen were collected from duodenum, antrum and esophagus. Apart from histopathological examination of biopsy material, rapid urease test (RUT) of the antral biopsy specimen and blood examination to estimate specific IgG ***antibodies*** to Hp by Indirect Solid Phase Enzyme ***Immunoassay*** was performed. The results of Hp IgG ***antibodies*** was compared with age matched controls. RESULTS: Thirty one children with RAP were subjected to endoscopic examination and their anti Hp IgG ***antibodies*** status compared with 26 controls. Hp colonization was detected in 7 children (23%) with RAP; by RUT in 23% and antral biopsy in 16% of cases. Anti Hp IgG ***antibodies*** were also positive in almost equal proportion (19%) of controls ($p = 0.757$). Endoscopic examination revealed esophagitis in 16% of cases and none had evidence of ***gastric*** or duodenal erosion, ulcer or cobblestone appearance of antrum. A significant correlation of Hp was noticed with chronic antral ***gastritis*** ($p = 0.002$), chronic duodenitis ($p = 0.02$) and age > 10 years ($p = 0.02$). No significant correlation was noticed between Hp colonization and various socioeconomic risk factors. CONCLUSION: Hp does not seem to be commonly associated with RAP in our patient population as Hp colonization was detected in only 23% of cases which was not significantly higher than the

seroprevalence of anti Hp IgG ***antibodies*** in the controls. However, a small sample size of our study limits drawing any firm conclusions. Antral ***gastritis*** and chronic duodenitis had a significant correlation with Hp colonization. RUT was found to be a reliable diagnostic test to detect Hp.

L11 ANSWER 95 OF 184 MEDLINE
AN 1998403644 MEDLINE
DN 98403644
TI Helicobacter ***pylori*** infection: an added stressor on iron status of women in the community.
AU Peach H G; Bath N E; Farish S J
CS University of Melbourne, Ballarat Health Services Base Hospital, VIC.. a.temperley@gpph.unimelb.edu.au
SO MEDICAL JOURNAL OF AUSTRALIA, (1998 Aug 17) 169 (4) 188-90. Journal code: M26. ISSN: 0025-729X.
CY Australia
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199811
EW 19981103
AB OBJECTIVE: To explore a possible association between Helicobacter ***pylori*** infection and iron status. DESIGN: Cross-sectional study. SETTING: Ballarat (a major regional city in Victoria), population 78000, October November 1997. PARTICIPANTS: 160 women and 152 men, a subsample of participants in a cardiovascular disease risk factor prevalence survey for whom frozen plasma was available. MAIN OUTCOME MEASURES: H. ***pylori*** IgG ***antibody*** status by enzyme ***immunoassay*** ; iron intake; plasma iron, transferrin and ferritin concentrations. RESULTS: 28% of women and 33% of men were infected with H. ***pylori*** . The mean (SEM) plasma ferritin concentration of infected women (59.3 [7.6] microg/L) was significantly lower than for non-infected women (88.8 [7.9] microg/L; P=0.002), after adjusting for age. Mean daily dietary iron intakes were similar in infected and non-infected women. CONCLUSIONS: H. ***pylori*** infection appears to be an additional stressor on women's iron status, but the mechanism remains to be determined.

L11 ANSWER 96 OF 184 MEDLINE
AN 1998081473 MEDLINE
DN 98081473
TI Comparison between a rapid office-based and ELISA serologic test in screening for Helicobacter ***pylori*** in children.
AU Elitsur Y; Neace C; Triest W E
CS Department of Pediatrics, Marshall University School of Medicine, Huntington, WV 25701-0195, USA.. yelitsur@musom.marshall.edu
SO HELICOBACTER, (1997 Dec) 2 (4) 180-4. Journal code: CY4. ISSN: 1083-4389.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199803
EW 19980304
AB BACKGROUND. The rapid diagnostic serological test for detection of

Helicobacter ***pylori*** (H. ***pylori***) infection in children has a significant advantage over the standard enzyme ***immunoassay*** (EIA) method, for its simplicity and rapid availability of results in a physician's office setting. We compared the immunochromatographic test with a standard enzyme ***immunoassay*** test in the pediatric population. MATERIALS AND METHODS. A retrospective analysis of 1147 serum samples from asymptomatic children and prospective analysis of 62 serum samples from symptomatic children undergoing diagnostic upper endoscopy were evaluated for the detection of H. ***pylori*** ***antibody*** by two commercially available serology tests. Each serum sample was tested by a rapid test (FlexSure HP, SmithKline Diagnostics, Inc.) and compared to the standard EIA method (HM-CAP, Enteric Products, Inc.). RESULTS. The rapid test, FlexSure HP, was comparable to the rapid EIA test in screening for H. ***pylori*** infection in symptomatic and asymptomatic children with sensitivity and specificity of 83-90% and 90-100%, respectively. Both methods had a comparable sensitivity and specificity for the detection of H. ***pylori*** -associated ***gastritis*** (60-70% and 94%, respectively). CONCLUSION. The rapid test is comparable to the standard EIA test and may be used by physicians in symptomatic children. The use of FlexSure HP as a screening tool for the prevalence of H. ***pylori*** infection in asymptomatic children may be limited by its low positive predictive value compared to the EIA method.

L11 ANSWER 97 OF 184 MEDLINE
AN 97268485 MEDLINE
DN 97268485
TI [The pathogenic role of Helicobacter ***pylori***].
O patogennoi roli Helicobacter ***pylori*** .
AU Ivashkin V T; Polozhentsev S D; Sultanov V K; Blinov V D; Solomakhin S V; Kretsu A P; Kalinin V K; Spesivtsev V N
SO TERAPEVTICHESKII ARKHIV, (1993) 65 (2) 11-3.
Journal code: VLU. ISSN: 0040-3660.
CY RUSSIA: Russian Federation
DT Journal; Article; (JOURNAL ARTICLE)
LA Russian
FS Priority Journals
EM 199707
EW 19970701
AB The urease test and bacterioscopy of impression smears were used to detect Helicobacter ***pylori*** (HP) in biopsies from pyloric ***gastric*** mucosa of 77 (89.5%) of 86 chronic ***gastritis*** patients, of 27 (4%) from 32 duodenal ulcer patients, of 84 (84.0%) from 100 healthy male subjects aged 18-20. There was focal hyperemia in pyloric part of the stomach in 59 patients, leukocytic infiltration of mucous membrane was found histologically in 78 ones. Close correlation between complaints, endoscopic and histological shifts, HP incidence rate was not registered. Positive results in determination of HP ***antibodies*** by enzyme ***immunoassay*** (EIA) were obtained in 32 (45.7%) of healthy subjects. EIA findings and histological evidence on HP presence failed to coincide in 42 (92.9%) duodenal ulcer patients.

L11 ANSWER 98 OF 184 MEDLINE
AN 97259999 MEDLINE
DN 97259999
TI Prevalence of Helicobacter ***pylori*** ***antibodies*** in

children in Bloemfontein, South Africa.
AU Pelser H H; Househam K C; Joubert G; van der Linde G; Kraaij P; Meinardi M; McLeod A; Anthony M
CS Department of Paediatrics and Child Health, University of the Orange Free State, Bloemfontein, South Africa.
SO JOURNAL OF PEDIATRIC GASTROENTEROLOGY AND NUTRITION, (1997 Feb) 24 (2) 135-9.
Journal code: JL6. ISSN: 0277-2116.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199709
EW 19970903
AB BACKGROUND: An association of H. ***pylori*** infection with chronic ***gastritis*** , peptic ulceration and ***gastric*** cancer is known. METHODS: Prevalence of IgG ***antibodies*** to Helicobacter ***pylori*** in children in the Bloemfontein, South Africa area was studied. Children attending the general pediatric outpatient department at Pelonomi Hospital in Bloemfontein were grouped according to age. A minimum of 100 children was investigated in each age group. Baseline demographic and socioeconomic data were collected. RESULTS: The study showed a high prevalence of H. ***pylori*** ***antibodies*** . Prevalence increased with age: 13.5% in children 3 months-2 years, 48.5% at 2-5 years, 67.3% at 5-10 years and 84.2% at 10-15 years. Investigation of the socioeconomic data in relation to the prevalence of H. ***pylori*** was inconclusive. CONCLUSIONS: This high prevalence needs further study.

L11 ANSWER 99 OF 184 MEDLINE
AN 93213900 MEDLINE
DN 93213900
TI [Evaluation of the cut-off point in the serological diagnosis of Helicobacter ***pylori*** infection in children using an enzyme ***immunoassay*** technique (letter)].
Valoracion del punto de corte (cut off) en el diagnostico serologico de la infeccion por Helicobacter ***pylori*** en ninos mediante una tecnica de enzimoinmunoanalisis.
AU Sanz J C; Martin E; Alarcon T; Martinez M J; Garcia-Novo M D; Lopez-Brea M
SO ENFERMEDADES INFECCIOSAS Y MICROBIOLOGIA CLINICA, (1993 Jan) 11 (1) 55.
Journal code: A10. ISSN: 0213-005X.
CY Spain
DT Letter
LA Spanish
EM 199307

L11 ANSWER 100 OF 184 MEDLINE
AN 91041503 MEDLINE
DN 91041503
TI [Detection of ***antibodies*** to Helicobacter ***pylori*** with the immunoenzyme test and indirect immunofluorescence].
Nachweis von Antikorpern gegen Helicobacter ***pylori*** mit Enzymimmuntest und indirekter Immunfluoreszenz.
AU Abb J; Striegel K; Fruhmorgen P
CS Mikrobiologisches Institut, Krankenanstalten Ludwigsburg..
SO LEBER, MAGEN, DARM, (1990 Sep) 20 (5) 224-30.

Journal code: L3P. ISSN: 0300-8622.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA German
FS Priority Journals
EM 199102
AB Sera from 56 adult patients were screened for the presence of IgG ***antibodies*** against Helicobacter ***pylori*** by enzyme ***immunoassay*** and indirect immunofluorescence. In addition, the detection of Helicobacter ***pylori*** in antral biopsy specimens was attempted by culture and histological methods. Colonisation of the antrum mucosa with Helicobacter ***pylori*** was observed in 39 of the 56 patients. IgG ***antibodies*** against Helicobacter ***pylori*** were detected by enzyme ***immunoassay*** in 34 of 39 infected patients. Thus, the enzyme ***immunoassay*** showed a sensitivity of 87.2 percent and a specificity of 82.4 percent. IgG ***antibodies*** against Helicobacter ***pylori*** were further detected by indirect immunofluorescence in 28 of 39 infected patients. Thus, indirect immunofluorescence showed a sensitivity of 66.7 percent and a specificity of 88.2 percent. Our results demonstrate that the enzyme ***immunoassay*** for IgG ***antibodies*** and other invasive or noninvasive methods for the detection of infection with Helicobacter ***pylori*** appear to be of equal sensitivity and specificity.

L11 ANSWER 101 OF 184 MEDLINE
AN 88257555 MEDLINE
DN 88257555
TI Immunoblot analysis of immune response to Campylobacter ***pylori*** and its clinical associations.
AU von Wulffen H; Grote H J; Gatermann S; Loning T; Berger B; Buhl C
CS Institut fur Medizinische Mikrobiologie und Immunologie, Universitatskrankenhaus Eppendorf, Hamburg, Federal Republic of Germany..
SO JOURNAL OF CLINICAL PATHOLOGY, (1988 Jun) 41 (6) 653-9.
Journal code: HT3. ISSN: 0021-9746.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 198810
AB Systemic immune response to Campylobacter ***pylori*** was detected by the immunoblot technique in serum samples from 200 patients, 129 blood donors, and 96 children. The results of the IgG immunoblot test showed excellent correlation with the detection of C ***pylori*** by culture and also with histopathological examination of the antrum, as well as with peptic ulcer disease. An IgA response also occurred and gave results comparable with those of the IgG immunoblot test, although on a quantitatively lower scale. The IgM immunoblots were of no help in the serodiagnosis of C ***pylori*** infection. The protein bands that seemed to be the most specific for C ***pylori*** and which were consistently observed in patients positive for C ***pylori*** were a 110 kilodalton and a 63 kilodalton band on the IgG immunoblot and an 89 kilodalton band on the IgA immunoblot. A 94 kilodalton and a 28 kilodalton band were also included in the evaluation. While immunoblot analysis may be used effectively for the serodiagnosis of C ***pylori*** infection and can distinguish between patients with normal antrum mucosa and those

with ***gastritis*** , the test does not help to distinguish between those patients with antrum ***gastritis*** who subsequently develop peptic ulcers and those who do not.

L11 ANSWER 102 OF 184 MEDLINE
AN 88149892 MEDLINE
DN 88149892
TI Campylobacter ***pylori*** in Swedish patients referred for gastroscopy.
AU Gnarpe H; Unge P; Blomqvist C; Makitalo S
CS Department of Clinical Bacteriology, Gavle Central Hospital, Sweden..
SO APMIS, (1988 Feb) 96 (2) 128-32.
Journal code: AMS. ISSN: 0903-4641.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 198806
AB Campylobacter ***pylori*** was isolated more often from patient with peptic ulcers and in an age-related manner in a material of 395 consecutive patients referred for gastroscopy. Direct microscopy was done with Gram and Acridine Orange stain and found too insensitive for practical use. All patients were investigated serologically with an enzyme ***immunoassay*** which showed excellent correlation with positive cultures for C. ***pylori*** . The findings were discussed, and the enzyme ***immunoassay*** , with a negative predictive value of 0.99, was found to be a valuable tool for primary screening of patients suspected of being carriers of C. ***pylori*** .

L11 ANSWER 103 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 2000:79951 SCISEARCH
GA The Genuine Article (R) Number: 276BQ
TI Seroepidemiology of Helicobacter ***pylori*** infection in a Jamaican community
AU Lindo J F; LynSue A E; Palmer C J; Lee M G; Vogel P; Robinson R D (Reprint)
CS UNIV W INDIES, DEPT LIFE SCI, KINGSTON 7, JAMAICA (Reprint); UNIV W INDIES, DEPT LIFE SCI, KINGSTON 7, JAMAICA; UNIV W INDIES, DEPT MICROBIOL, KINGSTON 7, JAMAICA; UNIV MIAMI, SCH MED, CTR DIS PREVENT, MIAMI, FL; UNIV W INDIES, DEPT MED, KINGSTON 7, JAMAICA
CYA JAMAICA; USA
SO TROPICAL MEDICINE & INTERNATIONAL HEALTH, (DEC 1999) Vol. 4, No. 12, pp. 862-866.
Publisher: BLACKWELL SCIENCE LTD, P O BOX 88, OSNEY MEAD, OXFORD OX2 0NE, OXON, ENGLAND.
ISSN: 1360-2276.
DT Article; Journal
FS CLIN
LA English
REC Reference Count: 40
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB We researched epidemiologic associations between environmental and demographic factors and prevalence of Helicobacter ***pylori*** infection in a suburban Jamaican community. Using a clustered sampling technique, 22 domestic yards enclosing 60 separate households were

randomly selected from a local community. All household members (n = 346) were invited to participate following informed consent; the overall compliance rate was 58.9%. A commercial enzyme ***immunoassay*** (HMaCAP) was used to detect IgG ***antibodies*** raised against H. ***pylori*** . Environmental and demographic information was obtained by questionnaire. The seroprevalence of H. ***pylori*** was 69.9% (n = 202). Analysis of the independent variables revealed three major components: Component 1 described, collectively, good personal hygiene and sanitation, indoor water supply and absence of straying animals in the peridomestic area; Component 2 included older age, good personal hygiene and large yard size; Component 3 the presence of domestic animals (cats and dogs) and, again, large yard size. These three complexes explained 42.2% of the variability in the data set. Logistic regression showed that Components 2 and 3 were independently associated with H. ***pylori*** seropositivity, indicating that a combination of demographic, environmental and zoonotic factors is involved in the spread of H. ***pylori*** infections at the tropical community level.

L11 ANSWER 104 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 1999:613049 SCISEARCH
GA The Genuine Article (R) Number: 222EP
TI Long-term follow-up study of serum immunoglobulin G and immunoglobulin A ***antibodies*** after Helicobacter ***pylori*** eradication
AU Kato S (Reprint); Furuyama N; Ozawa K; Ohnuma K; Iinuma K
CS TOHOKU UNIV, SCH MED, DEPT PEDIAT, AOBA KU, 1-1 SEIRYO MACHI, SENDAI, MIYAGI 9808574, JAPAN (Reprint); SENDAI CITY HOSP, DEPT PEDIAT, SENDAI, MIYAGI, JAPAN
CYA JAPAN
SO PEDIATRICS, (AUG 1999) Vol. 104, No. 2, Part 1, pp. E221-E225.
Publisher: AMER ACAD PEDIATRICS, 141 NORTH-WEST POINT BLVD, ELK GROVE VILLAGE, IL 60007-1098.
ISSN: 0031-4005.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 25
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Objective. There have been few studies concerning serum titers of anti-Helicobacter ***pylori*** immunoglobulin G (IgG) ***antibody*** >12 months after eradication of the original infection. Moreover, clinical usefulness of immunoglobulin A (IgA) ***antibody*** levels remains to be established. The purpose of this study was to investigate long-term responses of serum IgG-specific and IgA-specific ***antibodies*** to H ***pylori*** in children after eradication therapy.
Study Design. A total of 34 children, 2 to 17 years of age (mean: 11.7 years) with H ***pylori*** -associated gastroduodenal disease received eradication therapy (proton pump inhibitor-based dual or triple regimens). Diagnoses included nodular ***gastritis*** (n = 8), ***gastric*** ulcer (n = 7), and duodenal ulcer (n = 19). Upper gastrointestinal endoscopy and biopsy were performed before the therapy and at 1 to 2 months' posttreatment. H ***pylori*** infection and eradication were defined by biopsy-based tests; eradication was successful in 28 patients and unsuccessful in 6. Pretreatment IgG was positive in 30 patients (88.2%), and the IgA was positive in 31 (91.2%), who were entered into this study (duration less than or equal to 24 months). Serum samples were

obtained before treatment and at 1, 3, 6, 12, 18, and 24 months' posttreatment. IgG and IgA ***antibodies*** were measured using commercial enzyme ***immunoassay*** kits (HM-CAP and PP-CAP; Enteric Products, Inc, New York, NY).

Results. Compared with pretreatment values, IgG and IgA ***antibodies*** significantly and steadily decreased at 1 through 24 months' posttreatment in successfully treated patients. A decrease in titer of the IgA class was significantly greater than that of the IgG class at 1 to 12 months' follow-up. There was no significant decrease in titer of either ***antibody*** in all but 2 patients with eradication failure. A greater than or equal to 30% decrease in titer of the IgA ***antibody*** at 6 months indicated eradication with sensitivity of 90.5% and specificity of 100%. For the IgG ***antibody*** , a 30% decrease at 12 months showed equal sensitivity and specificity. Seroreversion rates of IgG and IgA ***antibodies*** were 53%; and 48% at 12 months and were 86% and 81% at 24 months, respectively. The mean periods from the completion of eradication therapy to seroreversion of IgG and IgA ***antibodies*** were 11.2 +/- 7.0 and 11.6 +/- 7.8 months, respectively (not significantly different). A higher pretreatment titer of Ige ***antibody*** was related to a longer period of seroreversion (r = 0.44). In one patient, C-13-urea breath test-confirmed reinfection was accompanied by reappearance of significant titers of the IgG and IgA ***antibodies*** .

Conclusions. A serology test is useful for evaluating eradication in children. Approximately half of patients with successful eradication remained to be IgG-seropositive and IgA-seropositive at 12 months' posttreatment When a decrease titer in ***antibody*** is used for assessing eradication, an endpoint of greater than or equal to 6 months is required. The IgA ***antibody*** may be a more convenient indicator of H ***pylori*** status than is the IgG ***antibody*** .

L11 ANSWER 105 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 1999:531148 SCISEARCH
GA The Genuine Article (R) Number: 212NW
TI Helicobacter ***pylori*** in the Canadian arctic: Seroprevalence and detection in community water samples
AU McKeown I; Orr P; Macdonald S; Kabani A; Brown R; Coghlan G; Dawood M; Embil J; Sargent M; Smart G; Bernstein C N (Reprint)
CS GB443 HLTH SCI CTR, GASTROENTEROL SECT, 820 SHERBROOK ST, WINNIPEG, MB R3A 1R9, CANADA (Reprint); UNIV MANITOBA, DEPT MED, WINNIPEG, MB, CANADA; UNIV MANITOBA, DEPT COMMUNITY HLTH SCI, WINNIPEG, MB R3T 2N2, CANADA; UNIV MANITOBA, DEPT MED MICROBIOL, WINNIPEG, MB, CANADA; NWT, KEEWATIN REG HLTH BOARD, WINNIPEG, MB, CANADA; CADHAM PROV LAB, RH RES LAB, WINNIPEG, MB, CANADA
CYA CANADA
SO AMERICAN JOURNAL OF GASTROENTEROLOGY, (JUL 1999) Vol. 94, No. 7, pp. 1823-1829.
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010.
ISSN: 0002-9270.
DT Article; Journal
FS CLIN
LA English
REC Reference Count: 25
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB OBJECTIVE: Many North American arctic communities are characterized by risk markers associated with Helicobacter ***pylori*** (H. ***pylori***) infection, including overcrowded housing and inadequate water supply and sanitation systems. Our aim was to determine the seroprevalence of H. ***pylori*** infection in two traditional Inuit communities in the central Canadian arctic and to test for the presence of H. ***pylori*** , by polymerase chain reaction (PCR), in local water supplies.

METHODS: Samples of venous whole blood from adults and capillary blood from children were collected and analyzed by enzyme ***immunoassay*** and Helisal Rapid Test, respectively, for IgG ***antibody*** to H. ***pylori*** . ***Antibodies*** to CagA were detected by enzyme ***immunoassay*** , and ABO and Lewis antigens were also determined. Demographic and clinical information were collected by questionnaire. Water samples from each community were tested for H. ***pylori*** by PCR.

RESULTS: One hundred-thirty (50.8%) of 256 subjects from the two communities were positive for H. ***pylori*** IgG ***antibodies*** . Seropositive subjects were more likely to be male, compared with seronegative individuals ($p = 0.01$). ***Antibody*** status did not differ with respect to age, community, alcohol or cigarette use, number of persons per household, gastrointestinal complaints or previous investigations, medications, or presence of blood group O, Lewis a-b+. CagA ***antibodies*** were detected in 78 (61.9%) of 126 H. ***pylori*** -seropositive subjects tested; however, 41 (35.3%) of 116 H. ***pylori*** -seronegative subjects were also CagA positive. Water samples taken from the water delivery truck in Chesterfield Inlet and two lakes near Repulse Bay were positive for H. ***pylori*** .

CONCLUSION: The seroprevalence of H. ***pylori*** in the study group was higher than rates in southern Canadian populations, but lower than the seroprevalence previously documented in a Canadian subarctic Indian (First Nations) community. The detection of H. ***pylori*** in local water supplies may indicate a natural reservoir for the organism or possible contamination from human sewage. (Am J Gastroenterol 1999;94:1823-1829. (C) 1999 by Am. Coll. of Gastroenterology).

L11 ANSWER 106 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 1998:719184 SCISEARCH
GA The Genuine Article (R) Number: 119LD
TI Evaluation of commercially available Helicobacter ***pylori*** serology kits: a review
AU Laheij R J F (Reprint); Straatman H; Jansen J B M J; Verbeek A L M
CS MIES 152, POB 9101, NL-6500 HB NIJMEGEN, NETHERLANDS (Reprint); UNIV NIJMEGEN HOSP, DEPT GASTROENTEROL, NL-6500 HB NIJMEGEN, NETHERLANDS; UNIV NIJMEGEN, DEPT EPIDEMIOL, NIJMEGEN, NETHERLANDS
CYA NETHERLANDS
SO JOURNAL OF CLINICAL MICROBIOLOGY, (OCT 1998) Vol. 36, No. 10, pp. 2803-2809.
Publisher: AMER SOC MICROBIOLOGY, 1325 MASSACHUSETTS AVENUE, NW, WASHINGTON, DC 20005-4171.
ISSN: 0095-1137.
DT General Review; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 90

L11 ANSWER 107 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 1998:699502 SCISEARCH
GA The Genuine Article (R) Number: 117NF
TI Analysis of immunoglobulin a ***antibodies*** to Helicobacter ***pylori*** in serum and ***gastric*** juice in relation to mucosal inflammation
AU Hayashi S (Reprint); Sugiyama T; Yokota K; Isogai H; Isogai E; Oguma K; Asaka M; Fujii N; Hirai Y
CS JICHI MED SCH, DEPT MICROBIOL, 3311-1 YAKUSHIJI, MINAMI KAWACHI, TOCHIGI 3290498, JAPAN (Reprint); HOKKAIDO UNIV, SCH MED, DEPT INTERNAL MED 3, SAPPORO, HOKKAIDO 060863, JAPAN; OKAYAMA UNIV, SCH MED, DEPT BACTERIOL, OKAYAMA 7008558, JAPAN; SAPPORO MED UNIV, SCH MED, DEPT MICROBIOL, SAPPORO, HOKKAIDO 060855, JAPAN; SAPPORO MED UNIV, SCH MED, ANIM EXPERIMENTAT CTR, SAPPORO, HOKKAIDO 060855, JAPAN
CYA JAPAN
SO CLINICAL AND DIAGNOSTIC LABORATORY IMMUNOLOGY, (SEP 1998) Vol. 5, No. 5, pp. 617-621.
Publisher: AMER SOC MICROBIOLOGY, 1325 MASSACHUSETTS AVENUE, NW, WASHINGTON, DC 20005-4171.
ISSN: 1071-412X.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 33
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Helicobacter ***pylori*** is a major etiologic agent in gastroduodenal disorders. In this study, immunoglobulin A (IgA) ***antibodies*** to H. ***pylori*** antigens were evaluated in serum and ***gastric*** juice specimens obtained from patients with ***gastritis*** or peptic ulcers by utilizing ***antibody*** capture enzyme-linked immunosorbent assays (ACELISAs). Urease alpha subunit (UA), urease beta subunit (UB), the 66-kDa heat shock protein (HSP), and the 25-kDa protein (25K) were used as antigens for the ACELISAs. The ***antibody*** titers of the ACELISAs reflect the ratio of H. ***pylori*** -specific IgA to total IgA. The ratio is stable, although the ***antibody*** concentration fluctuates in ***gastric*** juice. By using ACELISAs it was possible to evaluate quantitatively not only serum IgA ***antibodies*** but also ***gastric*** juice secretory IgA (S-IgA) ***antibodies*** . In both serum IgA and ***gastric*** juice S-IgA ACELISAs, the titers of ***antibody*** to HSP and 25K were remarkably correlated with the histologic grade of ***gastritis*** , whereas those to UA and UB were not strongly correlated with histologic grade. Thus, it is useful for estimating the histologic grade of ***gastritis*** to quantify serum IgA and ***gastric*** juice S-IgA ***antibodies*** to HSP and 25K.

L11 ANSWER 108 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 97:774500 SCISEARCH
GA The Genuine Article (R) Number: YB027
TI Helicobacter ***pylori*** Lewis expression is related to the host Lewis phenotype
AU Wirth H P (Reprint); Yang M Q; Peek R M; Tham K T; Blaser M J
CS UNIV ZURICH, SCH MED, DIV GASTROENTEROL, RAMISTR 100, CH-8091 ZURICH, SWITZERLAND (Reprint); VANDERBILT UNIV, SCH MED, DIV INFECT DIS,

NASHVILLE, TN 37212; VANDERBILT UNIV, SCH MED, DIV GASTROENTEROL, NASHVILLE, TN 37212; DEPT VET AFFAIRS MED CTR, NASHVILLE, TN 37212
CYA SWITZERLAND; USA
SO GASTROENTEROLOGY, (OCT 1997) Vol. 113, No. 4, pp. 1091-1098.
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399.
ISSN: 0016-5085.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 35
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Background & Aims: Lewis antigens occur in human ***gastric*** epithelium and in Helicobacter ***pylori*** lipopolysaccharide; their expression is polymorphic in both. Autoimmune mechanisms induced by bacterial Lewis expression have been proposed to cause ***gastritis*** . The aim of this study was to examine the relationship between bacterial and host ***gastric*** Lewis expression, as determined by the erythrocyte Lewis(a/b) phenotype, and between ***gastric*** histopathology and bacterial Lewis expression. Methods: H. ***pylori*** Lewis expression was determined by enzyme ***immunoassays*** , erythrocyte Lewis phenotype was assessed by agglutination tests, and ***gastric*** histopathology was scored blindly. Results: The host Lewis phenotype was (a+b-) in 15, (a-b+) in 34, and (a-b-) in 17 patients, therefore expressing Lewis x, y, or neither as their major ***gastric*** epithelial Lewis type 2 antigen. H. ***pylori*** from patients with Lewis(a+b-) expressed Lewis x more than y (1147 +/- 143 vs. 467 +/- 128 optical density units [ODU]; P = 0.006), isolates from patients with Lewis(a-b+) expressed Lewis x less than y (359 +/- 81 vs. 838 +/- 96 ODU; P = 0.0001), and isolates from Lewis(a-b-) patients expressed Lewis x and y approximately equally. ***Gastritis*** was unrelated to H. ***pylori*** Lewis expression. Conclusions: In mimicking host ***gastric*** epithelium, H. ***pylori*** cells not only express Lewis x and y, but the relative proportion of expression corresponds to the host Lewis phenotype, suggesting selection for host-adapted organisms.

L11 ANSWER 109 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 97:737204 SCISEARCH
GA The Genuine Article (R) Number: XY551
TI Helicobacter ***pylori*** infection in an Australian regional city: prevalence and risk factors
AU Peach H G (Reprint); Pearce D C; Farish S J
CS UNIV MELBOURNE, DEPT PUBL HLTH & COMMUNITY MED, BALLARAT HLTH SERV BASE HOSP, POB 577, BALLARAT, VIC 3353, AUSTRALIA (Reprint); UNIV MELBOURNE, EPIDEMIOL & BIOSTAT UNIT, DEPT PUBL HLTH & COMMUNITY MED, PARKVILLE, VIC 3052, AUSTRALIA
CYA AUSTRALIA
SO MEDICAL JOURNAL OF AUSTRALIA, (15 SEP 1997) Vol. 167, No. 6, pp. 310-313.
Publisher: AUSTRALASIAN MED PUBL CO LTD, LEVEL 1, 76 BERRY ST, SYDNEY NSW 2060, AUSTRALIA.
ISSN: 0025-729X.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 23

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Objective: To investigate the prevalence of Helicobacter ***pylori*** infection and potential risk factors for infection in an adult Australian population.

Design: Cross-sectional study.

Setting: Ballarat, a major regional city in Victoria (population, 78 000; 92% born in Australia), November 1994 to July 1995.

Participants: 217 adults randomly selected from the electoral roll.

Main outcome measures: H. ***pylori*** IgG ***antibody*** status by enzyme ***immunoassay*** ; amount of dental plaque; sociodemographic and other potential risk factors; odds ratios for risk factors determined by logistic regression analysis.

Results: Age-standardised prevalence of H. ***pylori*** infection was 30.6%. After adjustment for age, sex and socioeconomic index, positive H. ***pylori*** status was significantly associated with increasing number of tooth surfaces with a high plaque score (odds ratio [OR], 1.7; 95% confidence interval [CI], 1.1-2.7), increasing number of years in a job with public contact (OR, 1.7; 95% CI, 1.3-2.3), blood group B antigen (OR, 3.1 95% CI, 1.1-9.1), and having lived in a household with more than six members during childhood (OR, 2.5; 95% CI, 1.1-5.5). Negative H. ***pylori*** status was significantly associated with increasing education, having ever lived on a farm, and having teeth scaled less than once a year.

Conclusions: H. ***pylori*** infection is common. Dental plaque may be a reservoir for H. ***pylori*** , which is probably transmitted by person-to-person contact, and blood group B antigen may predispose to infection. Community education about effective oral hygiene and adoption of good hygiene practices by those with regular public contact may be important to prevent acquisition and transmission of H. ***pylori*** .

L11 ANSWER 110 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)

AN 97:379884 SCISEARCH

GA The Genuine Article (R) Number: WY184

TI Evaluation of a rapid, new method for detecting serum IgG ***antibodies*** to Helicobacter ***pylori***

AU Sharma T K; Young E L; Miller S; Cutler A F (Reprint)

CS SINAI HOSP, GASTROENTEROL SECT, 6767 W OUTER DR, DETROIT, MI 48235 (Reprint); SINAI HOSP, GASTROENTEROL SECT, DETROIT, MI 48235

CYA USA

SO CLINICAL CHEMISTRY, (MAY 1997) Vol. 43, No. 5, pp. 832-836.
Publisher: AMER ASSOC CLINICAL CHEMISTRY, 2101 L STREET NW, SUITE 202, WASHINGTON, DC 20037-1526.
ISSN: 0009-9147.

DT Article; Journal

FS LIFE

LA English

REC Reference Count: 26

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB There is an increased need for rapid, inexpensive tests to diagnose Helicobacter ***pylori*** infection. Our objective was to determine the performance characteristics of an immunochromatographic test (ICT) for detection of anti-H. ***pylori*** IgG ***antibodies*** . A commercially available ICT kit. (FlexSure(R) HP) was tested with a well-characterized cohort of banked sera as well as with fresh serum from randomly selected symptomatic patients. The ICT was evaluated with 107

stored sera and 96 prospective patients. The test correctly identified 65 of 68 H. ***pylori*** -infected and 37 of 39 noninfected stored sera and 54 of 57 infected and 30 of 39 noninfected patients. Sensitivity, specificity, and positive and negative predictive values were 96%, 95%, 97%, and 93% in stored serum and 95%, 77%, 86%, and 91% in fresh serum, respectively. We concluded that the ICT, reported at 4 min, is highly sensitive for detecting anti-H. ***pylori*** IgG ***antibodies*** in human serum. With a high negative predictive value, the test may be used to exclude H. ***pylori*** infection in symptomatic patients.

L11 ANSWER 111 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 97:137683 SCISEARCH
GA The Genuine Article (R) Number: WG312
TI Risk of infection with Helicobacter ***pylori*** and hepatitis A virus in different groups of hospital workers
AU Rudi J (Reprint); Toppe H; Marx N; Zuna I; Theilmann L; Stremmel W; Raedsch R
CS UNIV HEIDELBERG, DEPT MED, DIV GASTROENTEROL, BERGHEIMER STR 58, D-69115 HEIDELBERG, GERMANY (Reprint); ST JOSEF HOSP, DEPT MED, DIV GASTROENTEROL, WIESBADEN, GERMANY; GERMAN CANC RES CTR, RES PROGRAM RADIOL DIAGNOST & THERAPY, D-6900 HEIDELBERG, GERMANY
CYA GERMANY
SO AMERICAN JOURNAL OF GASTROENTEROLOGY, (FEB 1997) Vol. 92, No. 2, pp. 258-262.
Publisher: WILLIAMS & WILKINS, 351 WEST CAMDEN ST, BALTIMORE, MD 21201-2436.
ISSN: 0002-9270.
DT Article; Journal
FS CLIN
LA English
REC Reference Count: 34
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Objectives: The purpose of this study was to determine whether different staff groups in an acute care hospital are at increased risk of acquiring Helicobacter ***pylori*** and hepatitis A virus infection. Methods: We examined staff members of an acute care hospital for serum ***antibodies*** to H. ***pylori*** IgG (n = 457) and to hepatitis A virus (n = 434). The staff members were assigned to three groups: 1) nonmedical staff (n = 110), 2) medical and nursing staff (n = 272), and 3) medical and nursing staff working in a gastroenterology and endoscopy unit (n = 75). Serum ***antibodies*** were measured by validated enzyme ***immunoassays*** . A questionnaire inquiring about medical and professional history, history of upper GI pain and ulcer, as well as about the use of nonsteroidal antiinflammatory drugs or medication for GI complaints and smoking habits was completed by each person. Results: The seroprevalence of H. ***pylori*** was 35.5% in group I, 34.6% in group II, and 24.0% in group III (not significant). The seroprevalence of N. ***pylori*** ***antibodies*** increased with age ($p < 0.001$), and ***antibodies*** were present more frequently in women than in men (36.2 vs 25.4%, $p < 0.05$). After adjustment for age, duration of experience and the number of years working in the gastroenterology or endoscopy unit did not increase H. ***pylori*** seropositivity. No significant association was found between H. ***pylori*** seropositivity and history of upper GI pain, ulcers, use of nonsteroidal anti-inflammatory drugs or medication for Gf complaints, or tobacco use. The prevalence of

hepatitis A ***antibodies*** was similiar in the three groups (group I, 26.4%; II, 26.5%; III, 21.7%; not significant). Cross-tabulation showed that 67 subjects (15.4%) were seropositive for both H. ***pylori*** and hepatitis A ($p < 0.001$) and that 245 (56.5%) were negative for both. Seventy-seven (17.7.%) and 45 (10.4%) were seropositive for only H. ***pylori*** and for only hepatitis A, respectively.. Conclusions: Occupational exposure to patients in an acute care hospital as well as to patients and to endoscopic procedures of a gastroenterology and endoscopy unit does not increase the rate of infection with H. ***pylori*** . The significant correlation between the seroprevalences of H. ***pylori*** and hepatitis A ***antibodies*** suggests fecal-oral transmission of H. ***pylori*** .

L11 ANSWER 112 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 97:39325 SCISEARCH
GA The Genuine Article (R) Number: WA283
TI Association between Helicobacter ***pylori*** infection and serum ***pepsinogen*** concentrations in gastroduodenal disease
AU Matsumoto K (Reprint); Konishi N; Ohshima M; Hiasa Y; Kimura E; Samori T
CS JAPAN CLIN LABS INC, DEV SECT, 5-16-26 MINAMISUITA, SUITA, OSAKA 564, JAPAN (Reprint); NARA MED UNIV, DEPT PATHOL 2, KASHIHARA, NARA 634, JAPAN
CYA JAPAN
SO JOURNAL OF CLINICAL PATHOLOGY, (DEC 1996) Vol. 49, No. 12, pp. 1005-1008. Publisher: BRITISH MED JOURNAL PUBL GROUP, BRITISH MED ASSOC HOUSE, TAVISTOCK SQUARE, LONDON, ENGLAND WC1H 9JR.
ISSN: 0021-9746.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 37
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Aim-To investigate the association between Helicobacter ***pylori*** infection and serum ***pepsinogen*** (PG)1 and 2 concentrations in various gastroduodenal diseases.
Methods-Serum PG1 and 2 concentrations and ***antibodies*** to H ***pylori*** were measured by enzyme linked immunosorbent assay (ELISA); ***gastric*** mucosal pH was assessed and urease activity in biopsy tissue was determined. A comparison of the ELISA and urease test results permitted division of the cases into positive, false positive, false negative and negative categories for control, ***gastritis*** , and ulcer groups.
Results-The ***gastric*** mucosal pH and serum PG2 in cases positive for H ***pylori*** were significantly increased in ulcer and ***gastritis*** cases compared with H ***pylori*** negative cases. Similar tendencies were observed for the false positive and false negative categories.
Conclusions-A positive ELISA reaction for ***antibodies*** and an increased serum PG2 concentration are reliable indicators of H ***pylori*** infection.

L11 ANSWER 113 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 96:183295 SCISEARCH
GA The Genuine Article (R) Number: TX796
TI QUANTITATIVE DETECTION OF SECRETORY IMMUNOGLOBULIN-A TO HELICOBACTER-***PYLORI*** IN ***GASTRIC*** -JUICE - ***ANTIBODY*** -CAPTURE

ENZYME-LINKED-IMMUNOSORBENT-ASSAY
AU HAYASHI S (Reprint); SUGIYAMA T; HISANO K; AWAKAWA T; KUROKAWA I; YACHI A; ISOGAI H; ISOGAI E; YOKOTA K; HIRAI Y; OGUMA K; FUJII N
CS SAPPORO MED UNIV, SCH MED, DEPT MICROBIOL, S 1 W 17, SAPPORO, HOKKAIDO 060, JAPAN (Reprint); SAPPORO MED UNIV, SCH MED, DIV LAB DIAG, SAPPORO, HOKKAIDO 060, JAPAN; SAPPORO MED UNIV, SCH MED, DEPT INTERNAL MED, SECT 1, SAPPORO, HOKKAIDO 060, JAPAN; SAPPORO MED UNIV, SCH MED, DIV ANIM EXPERIMENTAT, SAPPORO, HOKKAIDO 060, JAPAN; HLTH SCI UNIV HOKKAIDO, SCH DENT, DEPT PREVENT DENT, ISHIKARI, HOKKAIDO, JAPAN; OKAYAMA UNIV, SCH MED, DEPT BACTERIOL, OKAYAMA 700, JAPAN
CYA JAPAN
SO JOURNAL OF CLINICAL LABORATORY ANALYSIS, (1996) Vol. 10, No. 2, pp. 74-77. ISSN: 0887-8013.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 18
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Helicobacter ***pylori*** is a major etiologic agent in gastroduodenal disorders. In this study, immunoglobulin A (IgA) ***antibodies*** to H. ***pylori*** were estimated in serum and ***gastric*** juice specimens from patients with ***gastritis*** and peptic ulcers using ***antibody*** capture enzyme-linked immunosorbent assays (ACELISAs). The ***antibody*** titers of the ACELISAs are independent of the ***antibody*** concentration and reflect the ratio of H. ***pylori*** -specific IgA to total IgA. The ratio is stable, although the ***antibody*** concentration fluctuates in ***gastric*** juice. Using the ACELISAs it was possible to evaluate quantitatively not only serum IgA (SR-IgA) ***antibodies*** but also secretory IgA (SC-IgA) ***antibodies*** in ***gastric*** juice. There were significant differences between the patients and control group in the SR-IgA and SC-IgA ACELISAs. Furthermore, the ACELISAs made it possible to compare between SR-IgA ***antibodies*** in serum and SC-IgA ***antibodies*** in ***gastric*** juice. In all patients, the ratios of H. ***pylori*** -specific IgA were higher in ***gastric*** juice than in serum. These results suggest that H. ***pylori*** SC-IgA ***antibodies*** are mainly produced by the local immune response in the ***gastric*** mucosa. Our studies indicate that ACELISA is well suited for the analysis of local immune response in mucosa. (C) 1996 Wiley-Liss, Inc.

L11 ANSWER 114 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 95:687270 SCISEARCH
GA The Genuine Article (R) Number: RX016
TI HELICOBACTER- ***PYLORI*** INFECTION IN FINNISH CHILDREN AND ADOLESCENTS - A SEROLOGIC CROSS-SECTIONAL AND FOLLOW-UP-STUDY
AU ASHORN M (Reprint); MAKI M; HALLSTROM M; UHARI M; AKERBLOM H K; VIIKARI J; MIETTINEN A
CS UNIV TAMPERE, DEPT CLIN MED, POB 607, SF-33101 TAMPERE, FINLAND (Reprint); TAMPERE UNIV HOSP, DEPT CLIN MED, TAMPERE, FINLAND; UNIV OULU, DEPT PEDIAT, SF-90100 OULU, FINLAND; UNIV HELSINKI, CHILDRENS HOSP, DEPT PEDIAT 2, HELSINKI, FINLAND; TURKU UNIV, DEPT MED, TURKU, FINLAND
CYA FINLAND
SO SCANDINAVIAN JOURNAL OF GASTROENTEROLOGY, (SEP 1995) Vol. 30, No. 9, pp. 876-879.

ISSN: 0036-5521.
DT Article; Journal
FS LIFE; CLIN
LA ENGLISH
REC Reference Count: 18
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Background: The purpose was to examine the epidemiology of Helicobacter ***pylori*** infection in Finnish children and adolescents. Methods: Blood samples taken from healthy subjects (n = 461) 3-18 years old were studied cross-sectionally for the presence of H. ***pylori*** ***antibodies*** . Additionally, blood samples drawn in 1980, 1983, 1986, and 1989 from 74 children born in 1977 were tested. Serum IgG-class ***antibodies*** to H. ***pylori*** were determined by an enzyme ***immunoassay*** . Results: In the cross-sectional series the mean ***antibody*** levels and the percentage of seropositive children increased with age. The overall seroprevalence was 10.2%. During the follow-up period from 3 to 12 pars of age the seropositivity increased from 4.6% to 5.7%. On the basis of the seroconversions between 3 and 12 years of age the annual incidence of H. ***pylori*** infection was calculated to be only 0.3%. Conclusions: In children seropositivity for H. ***pylori*** of the IgG class is often a sign of an infection acquired in early childhood. It seems likely that the age-dependent increase in the seropositivity reflects cumulation of a chronic infection.

L11 ANSWER 115 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 95:549348 SCISEARCH
GA The Genuine Article (R) Number: RN498
TI HELICOBACTER- ***PYLORI*** SEROPOSITIVITY AMONG SWEDISH ADULTS WITH AND WITHOUT ABDOMINAL SYMPTOMS - A POPULATION-BASED EPIDEMIOLOGIC-STUDY
AU AGREUS L (Reprint); ENGSTRAND L; SVARDSUDD K; NYREN O; TIBBLIN G
CS VARDCENTRALEN, S-74221 OSTHAMMAR, SWEDEN (Reprint); UNIV UPPSALA, DEPT FAMILY MED, CLIN EPIDEMIOL UNIT, S-75105 UPPSALA, SWEDEN; UNIV UPPSALA, DEPT CLIN MICROBIOL, S-75105 UPPSALA, SWEDEN; UNIV UPPSALA HOSP, DEPT CANC EPIDEMIOL, UPPSALA, SWEDEN; PRIMARY HLTH CARE CTR, OSTHAMMAR, SWEDEN
CYA SWEDEN
SO SCANDINAVIAN JOURNAL OF GASTROENTEROLOGY, (AUG 1995) Vol. 30, No. 8, pp. 752-757.
ISSN: 0085-5928.
DT Article; Journal
FS LIFE; CLIN
LA ENGLISH
REC Reference Count: 25
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Background: The role of Helicobacter ***pylori*** in functional dyspepsia is unclear. The aim of this population-based study was to determine whether the prevalence of H. ***pylori*** infection is higher among people with dyspepsia or irritable bowel syndrome (IBS) than among symptomless persons after control for age, sex, and socioeconomic status. Methods: In a postal questionnaire we asked a representative sample (20-79 years; n = 1260) from a Swedish municipality about abdominal symptoms in the preceding 3 months. A randomly selected subsample, 50 with dyspepsia, 50 with IBS, and 50 symptomless, matched with regard to age, sex, and education, were tested for the presence of IgG ***antibodies*** to H. ***pylori*** , using the HM-CAP ***immunoassay*** . Results: Fifty-five persons (38%) were H. ***pylori*** -seropositive. The

seroprevalence among dyspeptics (33%) did not exceed that in healthy people (48%) or in those reporting IBS (33%). The prevalence increased with age and with lower social class, but the latter association disappeared when age was taken into account. Neither sex nor symptom intensity predicted Helicobacter seropositivity. Conclusion: Our data are incompatible with an important aetiologic role for H. ***pylori*** in functional dyspepsia.

L11 ANSWER 116 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 94:770438 SCISEARCH
GA The Genuine Article (R) Number: PV007
TI THE PREVALENCE OF HELICOBACTER- ***PYLORI*** POSITIVITY IN HUMAN IMMUNODEFICIENCY VIRUS-INFECTED CHILDREN
AU BLECKER U (Reprint); KEYMOLEN K; LANCIERS S; BAHWERE P; SOUAYAH H; LEVY J; VANDENPLAS Y
CS FREE UNIV BRUSSELS, ACAD CHILDRENS HOSP, DEPT PEDIAT GASTROENTEROL, LAABEEKLAAU 101, B-1090 BRUSSELS, BELGIUM (Reprint); FREE UNIV BRUSSELS, ACAD CHILDRENS HOSP, DEPT ANESTHESIOL, B-1090 BRUSSELS, BELGIUM; HOP UNIV ST PIERRE, DEPT PEDIAT, BRUSSELS, BELGIUM
CYA BELGIUM
SO JOURNAL OF PEDIATRIC GASTROENTEROLOGY AND NUTRITION, (NOV 1994) Vol. 19, No. 4, pp. 417-420.
ISSN: 0277-2116.
DT Article; Journal
FS LIFE; CLIN
LA ENGLISH
REC Reference Count: 13
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB To investigate the prevalence of Helicobacter ***pylori*** infection in pediatric patients infected with the human immunodeficiency virus, we sought to detect the presence of ***antibodies*** against this organism in 23 human immunodeficiency virus-infected children of central African ethnic origin by means of a second-generation enzyme-linked ***immunoassay*** (ELISA) test for the detection of immunoglobulin G (IgG) ***antibodies*** to Helicobacter ***pylori*** (Malakit Helicobacter ***pylori*** , Biolab, Limal, Belgium). They were compared to an asymptomatic control population matched for age and ethnic origin. Blood samples were taken during routine blood analysis before the monthly administration of intravenous gamma-globulins in the human immunodeficiency virus-infected patients and during preoperative blood analysis in the control population. Despite the fact that most human immunodeficiency virus-infected patients had IgG ***antibodies*** against other frequently encountered pathogens, none of them had a positive serology for Helicobacter ***pylori*** , compared to 10 of 52 patients (19.2%) in the control population. This difference is statistically significant (p = 0.01).

L11 ANSWER 117 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 94:743762 SCISEARCH
GA The Genuine Article (R) Number: PT564
TI SERUM ***ANTIBODY*** -RESPONSE TO THE SUPERFICIAL AND RELEASED COMPONENTS OF HELICOBACTER- ***PYLORI***
AU BAZILLOU M; FENDRI C; CASTEL O; INGRAND P; FAUCHERE J L (Reprint)
CS CHU LA MILETRIE, MICROBIOL LAB A, BP 577, F-86021 POITIERS, FRANCE (Reprint); CHU LA MILETRIE, MICROBIOL LAB A, F-86021 POITIERS, FRANCE; FAC

MED & PHARM POITIERS, DEPT PEDAG & INFORMAT MED, F-86031 POITIERS, FRANCE; HOSP LA RABTA, MICROBIOL LAB, TUNIS, TUNISIA
CYA FRANCE; TUNISIA
SO CLINICAL AND DIAGNOSTIC LABORATORY IMMUNOLOGY, (MAY 1994) Vol. 1, No. 3, pp. 310-317.
ISSN: 1071-412X.
DT Article; Journal
FS CLIN
LA ENGLISH
REC Reference Count: 32
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Superficial and released components were extracted from six selected Helicobacter ***pylori*** strains. The protein and antigenic profiles of these extracts were representative of the profiles found most frequently among the clinical strains and included major peptidic fractions at 19, 23.5, 57, 68, 76, 118, and 132 kDa and major antigens at 68, 57, and 23.5 kDa. Immuno-cross-reactions were seen,vith a hyperimmune rabbit serum to Campylobacter fetus but not with sera to Campylobacter jejuni or Salmonella spp. An antigenic preparation was obtained by pooling equivalent quantities of each extract, and the antigenic preparation,vas used to study the ***antibody*** responses of sera from 65 French patients and 127 Tunisian patients. By enzyme-linked immunosorbent assay, we observed that the sera from French and Tunisian patients clustered into two populations, defined as ***antibody*** positive (72 patients) and ***antibody*** negative (120 patients). The ***antibody*** -positive patients were more frequently infected with H. ***pylori*** ($P < 0.01$) and were more frequently affected with ***gastritis*** ($P = 0.05$). However, no correlation between ***antibody*** levels and clinical signs of dyspepsia was noticed. The proportions of ***antibody*** -positive patients were similar in France and Tunisia. ***Antibody*** -positive and ***antibody*** -negative sera were studied by Western blot (immunoblot) analysis. The ***antibody*** -positive sera revealed an average of 7.7 antigenic bands, whereas the ***antibody*** -negative sera revealed an average of 2.4 antigenic bands ($P < 0.01$). The antigens between 15 and 40 kDa and greater than 66 kDa were specifically recognized by the ***antibody*** -positive sera, although in this molecular size range the ***antibody*** profiles of these sera exhibited a fairly high degree of diversity. We conclude that the superficial and released components from H. ***pylori*** contain a variety of bacterial immunogens and mag be useful in antigenic preparations for the serodiagnosis of H. ***pylori*** infections. Moreover, a group of antigens in combination appears to be useful for discriminating ***antibody*** -positive and ***antibody*** -negative patients.

L11 ANSWER 118 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 93:634261 SCISEARCH
GA The Genuine Article (R) Number: MB444
TI HELICOBACTER- ***PYLORI*** INFECTION IN VARIOUS GROUPS OF GENERAL-HOSPITAL INPATIENTS AND DONORS
AU KALININ A V (Reprint); SPESIVTSEV V N; SKVORTSOV S V; LYTSAR B N
CS NN BURDENKO CENT MIL CLIN HOSP, MOSCOW, RUSSIA (Reprint)
CYA RUSSIA
SO KLINICHESKAYA MEDITSINA, (1993) Vol. 71, No. 3, pp. 38-39.
ISSN: 0023-2149.
DT Article; Journal

FS CLIN
LA Russian
REC Reference Count: 8
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Incidence of Helicobacter ***pylori*** (HP) infection in inpatients of a general hospital and donors was evaluated immunologically: the serum was examined for IgG ***antibodies*** to HP using enzyme ***immunoassay*** . Of 354 examinees 89 had duodenal ulcer (DU), 101 were healthy donors. HP infection was found prevalent in all the groups of the inpatients. Cases of HP were more numerous in the gastroenterological department as compared to other departments (81.6% against 39.6%, respectively). Among patients of the gastroenterological department DU patients were most frequent HP carriers (93.2%), this being indicative of a close correlation between DU and HP revealed previously by other diagnosic methods. Rare occurrence of HP in normal subjects (6-11%) reported by many authors was not confirmed, as HP was detected in 33% of the donors. Age-related analysis points to early onset of HP infection (25-26% incidence in 18-20-year-olds), its peak in middle-aged and presenile patients (78-80% in 50-69-year-olds) and lower occurrence in senile patients (25% at the age over 70).

L11 ANSWER 119 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 93:629733 SCISEARCH
GA The Genuine Article (R) Number: MB612
TI PERFORMANCE OF HELICOBACTER- ***PYLORI*** ACID EXTRACT AND UREASE ENZYME-LINKED IMMUNOSORBENT ASSAYS IN RELATION TO C-14 UREA BREATH TEST
AU VONWULFFEN H (Reprint); GATERMANN S; WINDLER E; GABBE E; HEINRICH H C
CS UNIV KRANKENHAUS EPPENDORF, INST MED MIKROBIOL & IMMUNOL, MARTINSTR 52, D-20246 HAMBURG, GERMANY (Reprint); MED UNIV LUBECK, INST MED MIKROBIOL, D-23562 LUBECK, GERMANY; UNIV KRANKENHAUS EPPENDORF, MED KERNKLIN, D-20246 HAMBURG, GERMANY; UNIV KRANKENHAUS EPPENDORF, MED BIOCHEM ABT, D-20246 HAMBURG, GERMANY
CYA GERMANY
SO ZENTRALBLATT FUR BAKTERIOLOGIE-INTERNATIONAL JOURNAL OF MEDICAL MICROBIOLOGY VIROLOGY PARASITOLOGY AND INFECTIOUS DISEASES, (SEP 1993) Vol. 280, No. 1-2, pp. 203-213.
ISSN: 0934-8840.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 22
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The C-14-urea breath test has been shown to be a reliable non-invasive method to detect the presence or absence of H. ***pylori*** infection. Alternatively, a number of techniques have been devised to detect circulating ***antibodies*** against H. ***pylori*** in serum, the most commonly used being enzyme-linked immunosorbent assays (ELISA). In the present study we compared the value of two ELISA antigen preparations, an acid glycine extract and a urease preparation, in relation to the results achieved in a C-14-urea breath test. Seventy-five gastroenterology outpatients were screened for the presence of H. ***pylori*** infection using the urea breath test. At the same time serum specimens were obtained. Thirty-seven patients had a positive breath test, i.e. they expired more than 2% of the oral C-14 test dose within 60 min. Using the breath test as reference, sensitivity and specificity for the acid extract

were 89.2% and 84.2% respectively, and for the urease ELISA 81.1% and 89.5%. Agreement between the two ELISAs was found in 82.7%, overall agreement between all three tests was observed in 77.3%. All three tests were found to be useful for monitoring therapy directed against H. ***pylori*** .

L11 ANSWER 120 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 93:320262 SCISEARCH
GA The Genuine Article (R) Number: LC040
TI USEFULNESS OF SEVERAL COMMERCIAL ENZYME-LINKED ***IMMUNOASSAYS*** FOR DETECTION OF HELICOBACTER- ***PYLORI*** INFECTION IN CLINICAL MEDICINE
AU LOFFELD R J L F (Reprint); VRIESE W T J; STOBBERINGH E E
CS ZIEKENHUIS DE HEEL, DEPT INTERNAL MED, POB 210, 1500 EE ZAANDAM, NETHERLANDS (Reprint)
CYA NETHERLANDS
SO EUROPEAN JOURNAL OF GASTROENTEROLOGY & HEPATOLOGY, (MAY 1993) Vol. 5, No. 5, pp. 333-337.
ISSN: 0954-691X.
DT Article; Journal
FS CLIN
LA ENGLISH
REC No References Keyed
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Objective: To study six commercial enzyme-linked immunosorbent assays (ELISA) for detection of Helicobacter ***pylori*** immunoglobulin (Ig)G ***antibodies*** .

Design: A comparison of different ELISAs in patients with known H. ***pylori*** status.

Patients: Patients who underwent endoscopy during which biopsy specimens were taken for detection of H. ***pylori*** via standard histologic and microbiologic techniques.

Results: Positive and negative predictive values for the different ELISAs are comparable, but major inter- and intra-assay variability was present.

Conclusion: Commercial assays should be tested with a local reference population in order to obtain the optimal cut-off value via a receiver operating characteristics curve. The use of the cut-off values provided by the manufacturers introduces difficulties with interpretation and is not valid.

L11 ANSWER 121 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 92:557059 SCISEARCH
GA The Genuine Article (R) Number: JN651
TI EFFECT OF ANTIMICROBIAL THERAPY ON THE SPECIFIC SEROLOGICAL RESPONSE TO HELICOBACTER- ***PYLORI*** INFECTION
AU GLUPCZYNSKI Y (Reprint); BURETTE A; GOOSSENS H; DEPREZ C; BUTZLER J P
CS BRUGMANN UNIV HOSP, DEPT CLIN MICROBIOL, 4 PL A VAN GEHUCHTEN, B-1020 BRUSSELS, BELGIUM (Reprint); NOUVELLE CLIN BASILIQUE, GASTROENTEROL UNIT, B-1080 BRUSSELS, BELGIUM; ST PIETERS HOSP, B-1000 BRUSSELS, BELGIUM; BRUGMANN UNIV HOSP, DEPT PATHOL, B-1020 BRUSSELS, BELGIUM; WHO, COLLABORATING CTR ENTER CAMPYLOBACTER, B-1000 BRUSSELS, BELGIUM
CYA BELGIUM
SO EUROPEAN JOURNAL OF CLINICAL MICROBIOLOGY & INFECTIOUS DISEASES, (JUL 1992) Vol. 11, No. 7, pp. 583-588.
ISSN: 0934-9723.

DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 33
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The systemic immune response to Helicobacter ***pylori*** was studied in 247 infected adult patients before antimicrobial therapy and at different intervals following therapy. Endoscopy with simultaneous collection of biopsies was performed in all patients immediately before treatment, 4 to 6 weeks after the end of therapy and 6 to 12 months later. A C-14-urea breath test was performed 3 to 6 months after the end of treatment. Biopsy specimens were cultured and examined histologically using Giemsa stain. Sera were tested for Helicobacter ***pylori*** IgG ***antibodies*** with a commercial enzyme ***immunoassay*** using species-specific antigens. Overall, Helicobacter ***pylori*** was eradicated in 120 patients while the other 1127 remained infected with the organism. The follow-up period ranged from 4 weeks to 33 months (mean 10.2 months). Pretreatment IgG levels did not differ significantly between the two groups of patients. Six weeks after the end of treatment a slight but definite decrease in the IgG ***antibody*** levels was seen irrespective of treatment success. In the 127 patients who remained Helicobacter ***pylori*** -positive, the level of IgG ***antibodies*** remained stable or increased with time. A continuous fall in ***antibody*** levels was observed following bacterial eradication in the other 120 patients, but the difference in ***antibody*** levels between treatment responders and nonresponders became significant only more than six months after the end of treatment (p = 0.001). Serological testing may be useful for monitoring the outcome of long-term treatment of Helicobacter ***pylori*** infection and obviate the need for endoscopy.

L11 ANSWER 122 OF 184 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 92:140270 SCISEARCH
GA The Genuine Article (R) Number: HG098
TI EFFECT OF ALCOHOL-CONSUMPTION ON THE RISK OF HELICOBACTER- ***PYLORI*** INFECTION
AU HOOKNIKANNE J (Reprint)
CS UNIV HELSINKI, DEPT BACTERIOL & IMMUNOL, HAARTMANINKATU 3, SF-00290 HELSINKI 29, FINLAND (Reprint)
CYA FINLAND
SO DIGESTION, (OCT 1991) Vol. 50, No. 2, pp. 92-98.
ISSN: 0012-2823.
DT Article; Journal
FS LIFE; CLIN
LA ENGLISH
REC Reference Count: 30
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB To investigate the effect of alcohol consumption on the risk of Helicobacter ***pylori*** infection, standardized questionnaires on drinking habits were used to interview 451 patients, whose H. ***pylori*** status was determined both by culture and serology. Reported alcohol consumption did not increase the risk of H. ***pylori*** infection (a 1.0 odds ratio, CI95 0.6-1.6). However, when the patients were divided into two age-groups, those under 35 years who reported to use alcohol seemed to have a slightly higher risk of H.

pylori infection (a 3.3 odds ratio CI95 0.9-12.2) compared to those over 35 years (a 1.0 odds ratio, CI95 0.5-2.2). This phenomenon did not reach statistical significance. The type of alcohol consumed did not affect the age-adjusted risk of H. ***pylori*** infection. If pathologically defined chronic ***gastritis*** was found, the risk for H. ***pylori*** was high (a 26.7 odds ratio, CI95 12.1-59.0, for those under 35 years, and a 12.8 odds ratio, CI95 6.7-24.3, for those over 35 years of age).

L11 ANSWER 123 OF 184 USPATFULL
AN 2001:33009 USPATFULL
TI Human myosin heavy chain-like proteins and method of detecting nucleic acid encoding said proteins
IN Bandman, Olga, Mountain View, CA, United States
Yue, Henry, Sunnyvale, CA, United States
Corley, Neil C., Mountain Vew, CA, United States
Shah, Purvi, Sunnyvale, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 6197512 20010306
AI US 1998-216619 19981217 (9)
RLI Division of Ser. No. US 1997-966318, filed on 7 Nov 1997, now patented, Pat. No. US 6001593
DT Utility
EXNAM Primary Examiner: Carlson, Karen Cochrane
LREP Incyte Pharmaceuticals, Inc
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 13 Drawing Figure(s); 13 Drawing Page(s)
LN.CNT 2334
AB The invention provides human myosin heavy chain-like proteins (MHCP) and polynucleotides which identify and encode MHCP. The invention also provides expression vectors, host cells, ***antibodies*** , agonists, and antagonists. The invention also provides methods for treating disorders associated with expression of MHCP.

L11 ANSWER 124 OF 184 USPATFULL
AN 2000:174620 USPATFULL
TI Histidine kinase
IN Biswas, Sanjoy, Paoli, PA, United States
Throup, John P, Royersford, PA, United States
Wallis, Nicola G, Wayne, PA, United States
Zalacain, Magdalena, West Chester, PA, United States
PA SmithKline Beecham Corporation, Philadelphia, PA, United States (U.S. corporation)
SmithKline Beecham p.l.c., United Kingdom (non-U.S. corporation)
PI US 6165992 20001226
AI US 1998-81689 19980520 (9)
PRAI US 1997-48347 19970530 (60)
DT Utility
EXNAM Primary Examiner: Priebe, Scott D.
LREP Gimmi, Edward R.; Deibert, Thomas S.; King, William T.
CLMN Number of Claims: 30
ECL Exemplary Claim: 1,16
DRWN No Drawings

LN.CNT 2877
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides Histidine kinase polypeptides and polynucleotides encoding Histidine kinase polypeptides and methods for producing such polypeptides by recombinant techniques. Also provided are methods for utilizing Histidine kinase polypeptides to screen for antibacterial compounds.

L11 ANSWER 125 OF 184 USPATFULL
AN 2000:174365 USPATFULL
TI Method of detecting bacterial infection
IN Fawcett, Paul Thomas, Rising Sun, MD, United States
PA The Nemours Foundation, Wilmington, DE, United States (U.S. corporation)
PI US 6165736 20001226
AI US 1998-123231 19980728 (9)
DT Utility
EXNAM Primary Examiner: Bui, Phuong T.
LREP McGuireWoods, LLP
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 1 Drawing Figure(s); 1 Drawing Page(s)
LN.CNT 1393
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of developing sensitive and discriminatory diagnostic procedures for detecting active bacterial infection in animals, especially humans, basically involves partially digesting the genomic DNA of the infecting bacterial pathogen into a generally large number of ideally random fragments and finding proteins encoded by those fragments which evoke a discriminating response to specimens from viably infected animals. Cloning techniques are used to cause the genes of the multitude of DNA fragments to produce proteins. Groups of proteins encoded by the genes of each fragment are separately tested for the ability to generate an immune response in certain specimens from animals known to have "viable infection", "convalescent infection" and "naive status" with respect to infection by the infecting bacterial pathogen. The protein groups which evoke positive immune responses to viably infected but no immune response to naive specimens are identified as "selectively responsive proteins". Similarly, selectively responsive proteins which are found to evoke no immune response from convalescent specimens are identified as "discriminatingly responsive proteins". These selectively and discriminatingly responsive protein groups can be cloned in magnitude and used to test unknown patients for status of infection.

The method is amenable for developing tests based upon non-invasively obtained specimens, such as peripherally-obtained blood samples. Moreover, rigorous mapping of the pathogen genome is not prerequisite for carrying out the development method. Consequently, the development method can be used to obtain diagnostic procedures particularly suitable for generating individually inexpensive bacterial infection assays capable for screening large scale patient populations.

L11 ANSWER 126 OF 184 USPATFULL
AN 2000:160787 USPATFULL
TI TagA gene and methods for detecting predisposition to peptic ulceration and ***gastric*** carcinoma

IN Cover, Timothy L., Nashville, TN, United States
Blaser, Martin J., Nashville, TN, United States
Kleanthous, Harry, Cambridge, MA, United States
Tummuru, Murali K. R., Nashville, TN, United States
PA Vanderbilt University, Nashville, TN, United States (U.S. corporation)
PI US 6153390 20001128
AI US 1999-259437 19990301 (9)
RLI Continuation of Ser. No. US 1998-34306, filed on 2 Mar 1998, now patented, Pat. No. US 5876943 which is a division of Ser. No. US 1994-316397, filed on 30 Sep 1994, now patented, Pat. No. US 5733740 which is a continuation-in-part of Ser. No. US 1993-53614, filed on 26 Apr 1993, now patented, Pat. No. US 5403924 which is a continuation-in-part of Ser. No. US 1992-959940, filed on 13 Oct 1992, now abandoned
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Needle & Rosenberg, P.C.
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 2842
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides an isolated nucleic acid encoding an approximately 120-128 kilodalton antigen of Helicobacter ***pylori*** , or an antigenic fragment thereof, wherein the antigen is associated with peptic ulceration. The present invention also provides methods of detecting the presence of a Helicobacter ***pylori*** strain possessing the 120-128 kilodalton antigen in a subject, comprising the steps of contacting an ***antibody*** -containing sample from the subject with a detectable amount of the tagA antigen or antigenic polypeptide of the present invention and detecting the binding of the antigen or fragment and the ***antibody*** . The detection of a strain expressing the TagA antigen is an indication of predisposition to peptic ulceration and ***gastric*** carcinoma. A mutant H. ***pylori*** not expressing a functional TagA antigen is also provided.

L11 ANSWER 127 OF 184 USPATFULL
AN 2000:149964 USPATFULL
TI Human lysophospholipase
IN Hillman, Jennifer L., Mountain View, CA, United States
Shah, Purvi, Sunnyvale, CA, United States
Corley, Neil C., Mountain View, CA, United States
Murry, Lynn E., Portola Valley, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 6143544 20001107
AI US 1997-878862 19970619 (8)
DT Utility
EXNAM Primary Examiner: Hendricks, Keith D.; Assistant Examiner: Mayhew, Bradley S.
LREP Billings, Lucy J.; Price, Leanne C.Incyte Pharmaceuticals, Inc.
CLMN Number of Claims: 9
ECL Exemplary Claim: 1

DRWN 5 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 2199
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a new human lysophospholipase (JHLP) and polynucleotides which identify and encode IHLP. The invention also provides expression vectors, host cells, agonists, ***antibodies*** and antagonists. The invention also provides methods for treating inflammation and disorders associated with expression of IHLP.

L11 ANSWER 128 OF 184 USPATFULL
AN 2000:145883 USPATFULL
TI ATP-dependent RNA helicase protein
IN Bandman, Olga, Mountain View, CA, United States
Guegler, Karl J., Menlo Park, CA, United States
Corley, Neil C., Mountain View, CA, United States
Shah, Purvi, Sunnyvale, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 6139837 20001031
AI US 1998-149934 19980909 (9)
RLI Division of Ser. No. US 1997-892256, filed on 11 Jul 1997, now patented, Pat. No. US 5888792
DT Utility
EXNAM Primary Examiner: Slobodyansky, Elizabeth
LREP Incyte Pharmaceuticals, Inc.
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN 14 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 2351
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a human ATP-dependent RNA helicase (ADRH-1) and polynucleotides which identify and encode ADRH-1. The invention also provides expression vectors, host cells, agonists, ***antibodies*** and antagonists. The invention also provides methods for treating disorders associated with expression of ADRH-1.

L11 ANSWER 129 OF 184 USPATFULL
AN 2000:142121 USPATFULL
TI Microbiological media for isolation and indentification of enteric pathogens such as E. coli and salmonella
IN Bochner, Barry, Alameda, CA, United States
PA Biolog, Inc., CA, United States (U.S. corporation)
PI US 6136554 20001024
AI US 1997-819452 19970317 (8)
RLI Continuation of Ser. No. US 1995-484960, filed on 7 Jun 1995
DT Utility
EXNAM Primary Examiner: Marx, Irene
LREP Medlen & Carroll, LLP
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 3394
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to methods and media for the growth, enrichment, isolation, and presumptive identification of enteric

pathogens such as E. coli 0157:H7 and Salmonella. In particular, the organisms commonly associated with gastrointestinal infections of humans and other animals are distinguished based on their growth, colonial morphology and color. The present invention is also directed to methods and media for the growth, enrichment, isolation and presumptive identification of enteric pathogens such as E. coli 0157:H7 and Salmonella isolated from food, water, dairy, and environmental samples.

L11 ANSWER 130 OF 184 USPATFULL
AN 2000:134726 USPATFULL
TI Helicobacter ***pylori*** proteins useful for vaccines and diagnostics
IN Covacci, Antonello, Vc.Provenzano, 8, 53100, Siena, Italy
Bugnoli, Massimo, V. del Pozzo, 38, 53035, Monteriggioni, Italy
Telford, John, Via Sambuco, 43, 53010, Monteriggioni, Italy
Macchia, Giovanni, Via Monte Velino 57, 67051 Avezzano (AQ), Italy
Rappuoli, Rino, Via Calamandrei, 39, 53010 Quercegrossa (SI), Italy
PI US 6130059 20001010
AI US 1995-466662 19950606 (8)
RLI Division of Ser. No. US 1994-256848, filed on 21 Oct 1994, now abandoned which is a continuation of Ser. No. WO 1993-EP472, filed on 2 Mar 1993
PRAI IT 1992-FI52 19920302
WO 1993-EP158 19930125
DT Utility
EXNAM Primary Examiner: Bui, Phuong T.
LREP Harbin, Alisa A.; Paintin, Francis A.; Blackburn, Robert P.
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 14 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 2756
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Helicobacter ***pylori*** is known to cause or be a cofactor in type B ***gastritis*** , peptic ulcers, and ***gastric*** tumors. In both developed and developing countries, a high percentage of people are infected with this bacterium. The present invention relates generally to certain H. ***pylori*** proteins, to the genes which express these proteins, and to the use of these proteins for diagnostic and vaccine applications. Specifically, molecular cloning, nucteotide, and amino acid sequences for the H. ***pylori*** cytotoxin (CT), the "Cytotoxin Associated Immunodominant" (CAI) antigen, and the heat shock protein (hsp60). are described herein.

L11 ANSWER 131 OF 184 USPATFULL
AN 2000:113750 USPATFULL
TI F.sub.0 ATP synthase subunit
IN Hillman, Jennifer L., Mountain View, CA, United States
Goli, Surya K., Sunnyvale, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 6110722 20000829
AI US 1998-66049 19980424 (9)
RLI Division of Ser. No. US 1997-815177, filed on 11 Mar 1997, now patented, Pat. No. US 5786150
DT Utility
EXNAM Primary Examiner: Patterson, Jr., Charles L.

LREP Hamlet-Cox, DianaIncyte Pharmaceuticals, Inc.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 1951
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a human ATP synthase subunit (ASYS) and polynucleotides which encode ASYS. The invention also provides expression vectors, host cells, agonists, antisense molecules, ***antibodies*** , or antagonists. The invention also provides methods for producing ASYS and for treating disorders associated with expression of ASYS.

L11 ANSWER 132 OF 184 USPATFULL
AN 2000:109968 USPATFULL
TI iceA gene and related methods
IN Miller, Geraldine G., Franklin, TN, United States
Peek, Jr., Richard M., Nashville, TN, United States
Thompson, Stuart A., Whites Creek, TN, United States
Blaser, Martin J., Nashville, TN, United States
PA Vanderbilt University, Nashville, TN, United States (U.S. corporation)
PI US 6107464 20000822
AI US 1999-413140 19991006 (9)
RLI Division of Ser. No. US 1998-60584, filed on 15 Apr 1998, now patented, Pat. No. US 6004354 which is a division of Ser. No. US 1996-650528, filed on 20 May 1996, now patented, Pat. No. US 5780278, issued on 14 Jul 1998
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Needle & Rosenberg, P.C.
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 2188
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A purified IceA protein of Helicobacter ***pylori*** is provided. The protein is expressed as either an IceA 1 or an IceA 2 variant. A purified polypeptide fragment of the IceA protein is also provided. An antigenic fragment of IceA is provided. An isolated nucleic acid that encodes an IceA protein of H. ***pylori*** is provided. A nucleic acid that encodes an IceA 1 variant and a nucleic acid that encodes an IceA 2 variant is also provided. Fragments of the iceA gene are provided. A method of detecting the presence of an ***antibody*** against H. ***pylori*** in a sample is provided. The method comprises the following steps: a) contacting the sample with a purified IceA protein of H. ***pylori*** or a H. ***pylori*** -specific fragment thereof; and b) detecting the binding of the ***antibody*** in the sample to the protein or fragment, the detection of binding indicating the presence in the sample of ***antibodies*** against H. ***pylori*** . A method of detecting the presence of an ***antibody*** against an ulcerative Helicobacter ***pylori*** strain in a sample is also provided.

L11 ANSWER 133 OF 184 USPATFULL

AN 2000:94860 USPATFULL
TI Human lysophospholipase
IN Hillman, Jennifer L., Mountain View, CA, United States
Shah, Purvi, Sunnyvale, CA, United States
Murry, Lynn E., Portola Valley, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 6093561 20000725
AI US 1998-216386 19981218 (9)
RLI Division of Ser. No. US 1998-22940, filed on 12 Feb 1998 which is a continuation-in-part of Ser. No. US 1997-844120, filed on 29 Apr 1997, now patented, Pat. No. US 5858756
DT Utility
EXNAM Primary Examiner: Achutamurthy, Ponnathapu; Assistant Examiner: Mayhew, Bradley S.
LREP Muenzen, Colette C.; Mohan-Peterson, SheelaIncyte Pharmaceuticals, Inc.
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 9 Drawing Page(s)
LN.CNT 2310
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a human lysophospholipase (NHLP) and polynucleotides which identify and encode NHLP. The invention also provides expression vectors, host cells, ***antibodies*** , agonists, and antagonists. The invention also provides methods for treating or preventing disorders associated with expression of NHLP.

L11 ANSWER 134 OF 184 USPATFULL
AN 2000:91766 USPATFULL
TI Helicobacter ***pylori*** proteins useful for vaccines and diagnostics
IN Covacci, Antonello, Siena, Italy
Bugnoli, Massimo, Monteriggioni, Italy
Telford, John, Monteriggioni, Italy
Macchia, Giovanni, Avezzano, Italy
Rappuoli, Rino, Quercegrossa, Italy
PA Chiron S.p.A., Siena, Italy (non-U.S. corporation)
PI US 6090611 20000718
AI US 1995-471491 19950606 (8)
RLI Division of Ser. No. US 256848
PRAI IT 1992-FI52 19920302
WO 1993-EP158 19930125
DT Utility
EXNAM Primary Examiner: Bui, Phoung T.
LREP Paintin, Francis; Harbin, Alisa A.; Blackburn, Robert P.
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 14 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 2776
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A cytotoxin associated immunodominant antigen and the nucleic acid encoding the antigen from Helicobacter ***pylori*** are described. This antigen was identified from the cytotoxin positive CCUG 17874 Helicobacter ***pylori*** strain, and both the antigen and the DNA encoding it have been sequenced. The antigen is a hydrophilic,

surface-exposed protein having a molecular weight of 120-132 kDa. The nucleic acid encoding the antigen may be incorporated into a vector for transformation of host cells for expression of the antigen. Both the DNA and the antigen can be used in assays for detection of disease or infection by Helicobacter ***pylori*** , and may find use in treating and preventing infection by Helicobacter ***pylori*** and the diseases associated with such infection.

L11 ANSWER 135 OF 184 USPATFULL
AN 2000:80564 USPATFULL
TI Helicobacter catalase nucleotide sequences, their production and use
IN Sugiyama, Tosiro, Hokkaido, Japan
Kawabata, Tomohisa, Osaka, Japan
Hirayasu, Kazunari, Osaka, Japan
Tanaka, Takumi, Hyogo, Japan
PA Wako Pure Chemical Industries, Ltd., Osaka, Japan (non-U.S. corporation)
PI US 6080556 20000627
AI US 1996-657868 19960531 (8)
PRAI JP 1995-136564 19950602
JP 1996-83512 19960405
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Conlin, David G.; Neuner, George W.Dike, Bronstein, Roberts & Cushman LLP
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN 11 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 1491
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are amino acid sequences of polypeptides reacting with ***antibodies*** to Helicobacter ***pylori*** (HP), DNAs coding therefor, vectors containing said DNAs, transformants containing said vectors, a method for preparing said polypeptides by cultivating said transformants, and anti-HP ***antibody*** assaying reagents and HP gene detecting reagents comprising said polypeptides, thereby enabling specific, quantitative inspection of HP.

L11 ANSWER 136 OF 184 USPATFULL
AN 2000:77222 USPATFULL
TI Helicobacter ***pylori*** proteins useful for vaccines and diagnostics
IN Covacci, Antonello, Siena, Italy
Bugnoli, Massimo, Monteriggioni, Italy
Telford, John, Monteriggioni, Italy
Macchia, Giovanni, Avezzano, Italy
Rappuoli, Rino, Quercegrossa, Italy
PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)
PI US 6077706 20000620
AI US 1995-470260 19950606 (8)
RLI Division of Ser. No. US 256848
PRAI IT 1992-FI52 19920302
WO 1993-EP158 19930125
DT Utility
EXNAM Primary Examiner: Stucker, Jeffrey; Assistant Examiner: Bui, Phuong T.

LREP Paintin, Francis A.; Harbin, Alisa A.; Blackburn, Robert P.
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN 14 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 2677
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Helicobacter ***pylori*** known to cause or be a cofactor in type B ***gastritis*** , peptic ulcers, and ***gastric*** tumors. In both developed and developing countries, a high percentage of people are infected with this bacterium. The present invention relates generally to certain H. ***pylori*** proteins, to the genes which express these proteins, and to the use of these proteins for diagnostic and vaccine applications. Specifically, molecular cloning, nucleotide, and amino add sequences for the H. ***pylori*** cytotoxin (CT), the "Cytotoxin Associated Immunodominant" (CAI) antigen, and the heat shock protein (hsp60) are described herein.

L11 ANSWER 137 OF 184 USPATFULL
AN 2000:67576 USPATFULL
TI In vitro test for Helicobacter ***pylori***
IN Cripps, Allan, Curtin, Australia
Witt, Campbell, Bicton, Australia
Clancy, Robert Llewellyn, Newcastle, Australia
Stiel, Daniel, East Lindfield, Australia
PA Provalis UK Limited, Deeside, United Kingdom (non-U.S. corporation)
PI US 6068985 20000530
WO 9322682 19931111
AI US 1995-325264 19950426 (8)
WO 1993-GB894 19930429
19950426 PCT 371 date
19950426 PCT 102(e) date
PRAI CA 1992-2067603 19920429
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Foley & Lardner
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 766
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Contemporary infection by Helicobacter ***pylori*** in a human or other mammal can be detected by detecting H. ***pylori*** -specific IgG ***antibody*** in saliva, or other mucous secretion, with an antigen preparation from H. ***pylori*** . Diagnosis depends on detection of the antigen- ***antibody*** complex. For improved reliability, the antigen preparation comprises H. ***pylori*** -derived components of about 265 kDa and about 340 kDa and is substantially free of an H. ***pylori*** -derived component of about 440 kDa. The antigen preparation may be immobilised onto a solid support such as a nitrocellulose strip.

L11 ANSWER 138 OF 184 USPATFULL
AN 2000:57536 USPATFULL
TI Compositions and methods relating to drug discovery and detection and

treatment of gastrointestinal diseases
IN Corthesy-Theulaz, Irene, Epalinges, Switzerland
PA Kieta Holding SA, St-Prex, Switzerland (non-U.S. corporation)
PI US 6060241 20000509
AI US 1997-834776 19970403 (8)
PRAI US 1996-14906 19960405 (60)
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Cooley Godward LLP
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 13 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 2585
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A poly-3-hydroxybutyrate metabolic pathway essential for Helicobacter ***pylori*** survival in a host is provided. A novel Helicobacter ***pylori*** Coenzyme A transferase (Hp CoA-t), thiolase and PHB synthase as well as methods for their preparation and use are provided. Hp CoA-t and thiolase polynucleotides and proteins are provided as well as detection and preparative methods using such molecules. Methods for the determination of a propensity to develop ***gastritis*** , peptic ulcer disease, or ***gastric*** cancer is provided for by detection methods. Methods are also provided for the use of Hp CoA-t, thiolase or PHB synthase proteins and fragments retaining enzymatic activity in the identification of potential drug candidates for the treatment of some types of ***gastric*** disease. Pharmaceutical compositions containing Hp CoA-t protein fragments, antisense nucleic acids or other inhibitors of Hp CoA-t, thiolase and PHB synthase as well as methods for their use in the treatment of some types of ***gastric*** disease are also provided.

L11 ANSWER 139 OF 184 USPATFULL
AN 2000:31028 USPATFULL
TI ATP synthase subunit homolog
IN Tang, Y. Tom, San Jose, CA, United States
Corley, Neil C., Mountain View, CA, United States
Guegler, Karl J., Menlo Park, CA, United States
Baughn, Mariah R., San Leandro, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 6036954 20000314
AI US 1999-373029 19990811 (9)
RLI Division of Ser. No. US 1998-154802, filed on 17 Sep 1998
DT Utility
EXNAM Primary Examiner: Achutamurthy, Ponnathapu; Assistant Examiner: Slobodyansky, Elizabeth
LREP Muenzen, Colette C.Incyte Pharmaceuticals, Inc.
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 2512
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a human ATP synthase subunit homolog (ASYNT) and polynucleotides which identify and encode ASYNT. The invention also

provides expression vectors, host cells, ***antibodies*** , agonists, and antagonists. The invention also provides methods for diagnosing, treating or preventing disorders associated with expression of ASYNT.

L11 ANSWER 140 OF 184 USPATFULL
AN 2000:1708 USPATFULL
TI Process for detecting a variant CD44 gene product
IN Ponta, Helmut, Linkenheim-Hochstetten, Germany, Federal Republic of
Heider, Karl-Heinz, Waldbronn-Reichenbach, Germany, Federal Republic of
Herrlich, Peter, Karlsruhe, Germany, Federal Republic of
Pals, Steven T., Amsterdam, Netherlands
Dall, Peter, Dusseldorf, Germany, Federal Republic of
PA Boehringer Ingelheim International GmbH, Germany, Federal Republic of (non-U.S. corporation)
PI US 6010865 20000104
WO 9500851 19950105
AI US 1996-564225 19960603 (8)
WO 1994-EP1952 19940615
19960603 PCT 371 date
19960603 PCT 102(e) date
PRAI DE 1993-4320624 19930622
DE 1993-4320623 19930622
DE 1993-4321944 19930702
DE 1994-4414787 19940428
DT Utility
EXNAM Primary Examiner: Hutzell, Paula K.; Assistant Examiner: Ungar, Susan
LREP Finnegan, Henderson, Farabow, Garrett & Dunner, L.L.P.
CLMN Number of Claims: 7
ECL Exemplary Claim: 1,3
DRWN 45 Drawing Figure(s); 16 Drawing Page(s)
LN.CNT 1294
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to a process for diagnosing and analysing tumours which is based on detecting the expression of certain variant exons of the CD44-gene. Detection may be carried out at the protein or nucleic acid level. In a preferred embodiment the expression is detected in biopsy material using exon-specific ***antibodies*** . Thus, for example, v6-expression is a suitable prognostic parameter for breast cancer, the expression of a transitional epitope which is coded by exons v7 and v8 serves to diagnose cervical cancer.

L11 ANSWER 141 OF 184 USPATFULL
AN 1999:167130 USPATFULL
TI Treatment and prevention of helicobacter infection
IN Doidge, Christopher V., Vincent, Australia
Lee, Adrian, Lane Cove, Australia
Radcliff, Flona J., Sydney, Australia
Hazell, Stuart L., Glenfield, Australia
PA The University of New South Wales, Kensington, Australia (non-U.S. corporation)
CSL Limited, Victoria, Australia (non-U.S. corporation)
PI US 6005090 19991221
AI US 1996-695987 19960815 (8)
RLI Continuation-in-part of Ser. No. WO 1995-AU335, filed on 8 Jun 1995
PRAI AU 1994-6124 19940608

DT Utility
EXNAM Primary Examiner: Chin, Christopher L.; Assistant Examiner: Portner, Ginny Allen
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1390
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An antigenic preparation for use in the treatment or prevention of Helicobacter infection in a mammalian host, comprises the catalase enzyme of Helicobacter bacteria, particularly the catalase enzyme of H. ***pylori*** or H. felis, or an immunogenic fragment thereof.

L11 ANSWER 142 OF 184 USPATFULL
AN 1999:166833 USPATFULL
TI Human lysophospholipase
IN Hillman, Jennifer L., Mountain View, CA, United States
Shah, Purvi, Sunnyvale, CA, United States
Corley, Neil C., Mountain View, CA, United States
Murry, Lynn E., Portola Valley, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 6004792 19991221
AI US 1998-216001 19981217 (9)
RLI Division of Ser. No. US 1997-878862, filed on 19 Jun 1997
DT Utility
EXNAM Primary Examiner: Achutamurthy, Ponnathapu; Assistant Examiner: Mayhew, Bradley S.
LREP Incyte Pharmaceuticals, Inc.
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 2205
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a new human lysophospholipase (IHLP) and polynucleotides which identify and encode IHLP. The invention also provides expression vectors, host cells, agonists, ***antibodies*** and antagonists. The invention also provides methods for treating inflammation and disorders associated with expression of IHLP.

L11 ANSWER 143 OF 184 USPATFULL
AN 1999:166395 USPATFULL
TI IceA gene and related methods
IN Miller, Geraldine G., Franklin, TN, United States
Peek, Jr., Richard M., Nashville, TN, United States
Thompson, Stuart A., Whites Creek, TN, United States
Blaser, Martin J., Nashville, TN, United States
PA Vanderbilt University, Nashville, TN, United States (U.S. corporation)
PI US 6004354 19991221
AI US 1998-60584 19980415 (9)
RLI Division of Ser. No. US 1996-650528, filed on 20 May 1996, now patented, Pat. No. US 5780278
DT Utility
EXNAM Primary Examiner: Chin, Christopher L.; Assistant Examiner: Portner, Ginny Allen

LREP Needle & Rosenberg
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 2180
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A purified IceA protein of Helicobacter ***pylori*** is provided. The protein is expressed as either an IceA 1 or an IceA 2 variant. A purified polypeptide fragment of the IceA protein is also provided. An antigenic fragment of IceA is provided. An isolated nucleic acid that encodes an IceA protein of H. ***pylori*** is provided. A nucleic acid that encodes an IceA 1 variant and a nucleic acid that encodes an IceA 2 variant is also provided. Fragments of the iceA gene are provided. A method of detecting the presence of an ***antibody*** against H. ***pylori*** in a sample is provided. The method comprises the following steps: a) contacting the sample with a purified IceA protein of H. ***pylori*** or a H. ***pylori*** -specific fragment thereof; and b) detecting the binding of the ***antibody*** in the sample to the protein or fragment, the detection of binding indicating the presence in the sample of ***antibodies*** against H. ***pylori*** . A method of detecting the presence of an ***antibody*** against an ulcerative Helicobacter ***pylori*** strain in a sample is also provided.

L11 ANSWER 144 OF 184 USPATFULL
AN 1999:163449 USPATFULL
TI Human myosin heavy chain-like proteins
IN Bandman, Olga, Mountain View, CA, United States
Yue, Henry, Sunnyvale, CA, United States
Corley, Neil C., Mountain View, CA, United States
Shah, Purvi, Sunnyvale, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 6001593 19991214
AI US 1997-966318 19971107 (8)
DT Utility
EXNAM Primary Examiner: Carlson, Karen Cochrane
LREP Incyte Pharmaceuticals, Inc.
CLMN Number of Claims: 8
ECL Exemplary Claim: 1
DRWN 13 Drawing Figure(s); 13 Drawing Page(s)
LN.CNT 2545
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides human myosin heavy chain-like proteins (MHCP) and polynucleotides which identify and encode MHCP. The invention also provides expression vectors, host cells, ***antibodies*** , agonists, and antagonists. The invention also provides methods for treating disorders associated with expression of MHCP.

L11 ANSWER 145 OF 184 USPATFULL
AN 1999:150966 USPATFULL
TI Human ankyrin family protein
IN Tang, Y. Tom, San Jose, CA, United States
Guegler, Karl J., Menlo Park, CA, United States
Corley, Neil C., Mountain View, CA, United States

Yue, Henry, Sunnyvale, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 5989863 19991123
AI US 1998-172977 19981014 (9)
DT Utility
EXNAM Primary Examiner: Campbell, Eggerton A.; Assistant Examiner: Srivastava, Devesa
LREP Incyte Pharmaceuticals, Inc.
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 2663
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a human ankyrin family protein (ANFP) and polynucleotides which identify and encode ANFP. The invention also provides expression vectors, host cells, ***antibodies*** , agonists, and antagonists. The invention also provides methods for diagnosing, treating, or preventing disorders associated with expression of ANFP.

L11 ANSWER 146 OF 184 USPATFULL
AN 1999:150943 USPATFULL
TI Estimation of active infection by heliobacter ***pylori***
IN D'Angelo, Joseph P., Miami, FL, United States
Zhe, Jin, Miami, FL, United States
PA Americare International Diagnostics, Inc., Miami, FL, United States (U.S. corporation)
PI US 5989840 19991123
AI US 1997-865089 19970529 (8)
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Lerner, Herbert L.; Greenberg, Laurence A.
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 586
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a diagnostic apparatus for estimating an active Heliobacter ***pylori*** infectious agent in saliva, comprising in combination an ***immunoassay*** chamber in which a first portion of said saliva is subjected to serological test for ***antibody*** to said infectious agent and a chemical reaction chamber in which a second portion of said saliva is subjected to chemical analysis for an ammonia constituent thereof.

L11 ANSWER 147 OF 184 USPATFULL
AN 1999:150925 USPATFULL
TI ATP synthase subunit homolog
IN Tang, Y. Tom, San Jose, CA, United States
Corley, Neil C., Mountain View, CA, United States
Guegler, Karl J., Menlo Park, CA, United States
Baughn, Mariah R., San Leandro, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)

PI US 5989822 19991123
AI US 1998-154802 19980917 (9)
DT Utility
EXNAM Primary Examiner: Achutamurthy, Ponnathapura; Assistant Examiner: Slobodyansky, Elizabeth
LREP Incyte Pharmaceuticals, Inc.; Muenzen, Colette C.
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 2446
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a human ATP synthase subunit homolog (ASYNT) and polynucleotides which identify and encode ASYNT. The invention also provides expression vectors, host cells, ***antibodies*** , agonists, and antagonists. The invention also provides methods for diagnosing, treating or preventing disorders associated with expression of ASYNT.

L11 ANSWER 148 OF 184 USPATFULL
AN 1999:146266 USPATFULL
TI Platinum-containing compounds, methods for their preparation and applications thereof
IN Houthoff, Hendrik J., Amsterdam, Netherlands
Reedijk, Jan, Leiden, Netherlands
Jelsma, Tinka, Almere, Netherlands
Van Es, Remco Maria, Zaan, Netherlands
van den Berg, Franciscus Michiel, Hoofddorp, Netherlands
Lempers, Edwin Leo Mario, Julianadorp, Netherlands
Bloemink, Marieke Johanna, Oegstgeest, Netherlands
PA Kreatech Diagnostics, Amsterdam, Netherlands (non-U.S. corporation)
PI US 5985566 19991116
AI US 1997-910070 19970812 (8)
RLI Continuation of Ser. No. US 1995-470265, filed on 6 Jun 1995, now patented, Pat. No. US 5714327, issued on 3 Feb 1998 which is a continuation-in-part of Ser. No. US 1993-975586, filed on 19 Oct 1993, now patented, Pat. No. US 5580990, issued on 3 Dec 1996
PRAI NL 1990-1639 19900719
DT Utility
EXNAM Primary Examiner: Degen, Nancy; Assistant Examiner: Sandals, William
LREP Hoffmann & Baron, LLP
CLMN Number of Claims: 15
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1534
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides platinum-based probe compounds having the structure: ##STR1## wherein: Pt is a platinum atom, PROBE is a probe biomolecule for associating to a target biomolecule, M is a detectable marker moiety, and X and Y are stabilizing substituents. Also provided are platinum-based labeling compounds having the structure: ##STR2## wherein: Pt is a platinum atom, M is a detectable marker moiety, A is a displaceable leaving group, and X and Y are stabilizing substituents. The invention further provides platinum-based linker compounds having the structure: ##STR3## wherein: Pt is a platinum atom, A and B are the same or different reactive moieties, and X and Y are stabilizing substituents. Other Pt.sup.II and Pt.sup.IV compounds are also provided.

Moreover, the invention provides methods for the preparation and use of these compounds, as well as diagnostic kits which contain the compounds.

L11 ANSWER 149 OF 184 USPATFULL
AN 1999:136950 USPATFULL
TI Human reticulocalbin isoforms
IN Bandman, Olga, Mountain View, CA, United States
Hillman, Jennifer L., Mountain View, CA, United States
Lal, Preeti, Santa Clara, CA, United States
Corley, Neil C., Mountain View, CA, United States
Shah, Purvi, Sunnyvale, CA, United States
PA Incyte Pharamceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 5976801 19991102
AI US 1997-910927 19970808 (8)
DT Utility
EXNAM Primary Examiner: Fredman, Jeffrey
LREP Price, Leanne C.Incyte Pharmaceuticals, Inc.
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 18 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 2648
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides two human reticulocalbin isoforms designated individually as RCN .gamma. and RCN .delta. and collectively as RCN, and polynucleotides which identify and encode RCN. The invention also provides expression vectors, host cells, agonists, ***antibodies*** and antagonists. The invention also provides methods for treating disorders associated with expression of RCN.

L11 ANSWER 150 OF 184 USPATFULL
AN 1999:124759 USPATFULL
TI Human lysophospholipase
IN Hillman, Jennifer L., Mountain View, CA, United States
Shah, Purvi, Sunnyvale, CA, United States
Murry, Lynn E., Portola Valley, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 5965423 19991012
AI US 1998-22940 19980212 (9)
RLI Continuation-in-part of Ser. No. US 1997-844120, filed on 29 Apr 1997
DT Utility
EXNAM Primary Examiner: Hendricks, Keith D.; Assistant Examiner: Mayhew, Bradley S.
LREP Mohan-Peterson, SheelaIncyte Pharmaceuticals, Inc.
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 9 Drawing Page(s)
LN.CNT 2264
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a human lysophospholipase (NHLP) and polynucleotides which identify and encode NHLP. The invention also provides expression vectors, host cells, ***antibodies*** , agonists, and antagonists. The invention also provides methods for treating or preventing disorders associated with expression of NHLP.

L11 ANSWER 151 OF 184 USPATFULL
AN 1999:121547 USPATFULL
TI ATP synthase Fo subunit
IN Hillman, Jennifer L., Mountain View, CA, United States
Goli, Surya K., Sunnyvale, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 5962646 19991005
AI US 1998-216625 19981216 (9)
RLI Division of Ser. No. US 1998-94080, filed on 9 Jun 1998 which is a division of Ser. No. US 1997-948195, filed on 9 Oct 1997, now patented, Pat. No. US 5763248 which is a continuation of Ser. No. US 1997-819395, filed on 17 Mar 1997, now abandoned
DT Utility
EXNAM Primary Examiner: Degen, Nancy; Assistant Examiner: Wang, Andrew
LREP Mohan-Peterson, SheelaIncyte Pharmaceuticals, Inc.
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1999
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a human ATP synthase d subunit (ASYSD) and polynucleotides which encode ASYSD. The invention also provides expression vectors, host cells, agonists, antisense molecules, ***antibodies*** , or antagonists. The invention also provides to methods for producing ASYSD and for treating disorders associated with expression of ASYSD.

L11 ANSWER 152 OF 184 USPATFULL
AN 1999:106303 USPATFULL
TI Method of screening for ATP synthase Fo subunit
IN Hillman, Jennifer L., Mountain View, CA, United States
Goli, Surya K., Sunnyvale, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 5948625 19990907
AI US 1998-94080 19980609 (9)
RLI Division of Ser. No. US 1997-948195, filed on 9 Oct 1997, now patented, Pat. No. US 5763248 which is a continuation of Ser. No. US 1997-819395, filed on 17 Mar 1997, now abandoned
DT Utility
EXNAM Primary Examiner: LeGuyader, John L.; Assistant Examiner: Wang, Andrew
LREP Mohan-Peterson, SheelaIncyte Pharmaceuticals, Inc.
CLMN Number of Claims: 4
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 1993
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a human ATP synthase d subunit (ASYSD) and polynucleotides which encode ASYSD. The invention also provides expression vectors, host cells, agonists, antisense molecules, ***antibodies*** , or antagonists. The invention also provides methods for producing ASYSD and for treating disorders associated with expression of ASYSD.

L11 ANSWER 153 OF 184 USPATFULL
AN 1999:89021 USPATFULL
TI Vacuolar ***atpase*** subunit AC45
IN Hillman, Jennifer L., Mountain View, CA, United States
Shah, Purvi, Sunnyvale, CA, United States
Corley, Neil C., Mountain View, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 5932444 19990803
AI US 1997-959011 19971028 (8)
DT Utility
EXNAM Primary Examiner: Huff, Sheela; Assistant Examiner: Eyler, Yvonne
LREP Incyte Pharmaceuticals, Inc.
CLMN Number of Claims: 8
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 2221
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a human vacuolar ***ATPase*** subunit AC45 (HAC45) and polynucleotides which identify and encode HAC45. The invention also provides expression vectors, host cells, agonists, ***antibodies*** , and antagonists. The invention also provides methods for treating or preventing disorders associated with expression of HAC45.

L11 ANSWER 154 OF 184 USPATFULL
AN 1999:89007 USPATFULL
TI ***Immunoassay*** for H. ***pylori*** in fecal specimens
IN Larka, Christopher Vance, Cincinnati, OH, United States
Yi, Ching Sui Arthur, Cincinnati, OH, United States
Kozak, Kenneth James, Cincinnati, OH, United States
PA Meridian Diagnostics, Inc., Cincinnati, OH, United States (U.S. corporation)
PI US 5932430 19990803
AI US 1998-37894 19980310 (9)
RLI Continuation-in-part of Ser. No. US 1997-897732, filed on 21 Jul 1997, now patented, Pat. No. US 5871942 which is a continuation-in-part of Ser. No. US 1996-647115, filed on 9 May 1996, now patented, Pat. No. US 5716791
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Thompson, Hine & Flory LLP
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 1 Drawing Page(s)
LN.CNT 616
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A process for the determination of H. ***pylori*** in a fecal specimen comprising (a) dispersing a fecal specimen suspected of carrying H. ***pylori*** in a sample diluent; (b) contacting the fecal specimen in the diluent with a first polyclonal ***antibody*** for H. ***pylori*** antigen to form a complex of the ***antibody*** and the antigen; (c) separating said specimen and said

complex; (d) exposing the complex to a second polyclonal ***antibody*** for said antigen and a portion of the ***antibody*** reacting with said complex, one of said first and second ***antibody*** being bound to a solid carrier and the other being labeled with a detection agent; and (e) determining the amount of the labeled ***antibody*** and in turn determining the presence of H. ***pylori*** antigen in said fecal specimen.

L11 ANSWER 155 OF 184 USPATFULL
AN 1999:85216 USPATFULL
TI Compositions comprising isolated Helicobacter ***pylori*** CagI polynucleotides and method of preparation thereof
IN Covacci, Antonello, Siena, Italy
PA Chiron S.p.A., Italy (non-U.S. corporation)
PI US 5928865 19990727
AI US 1995-477451 19950607 (8)
RLI Continuation-in-part of Ser. No. US 1995-425194, filed on 20 Apr 1995, now abandoned And Ser. No. US 1995-471491, filed on 6 Jun 1995 which is a division of Ser. No. US 256848
PRAI IT 1992-FI52 19920302
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Woodcock, Washburn, Kurtz, Mackiewicz & Norris; Harbin, Alisa A.; Blackburn, Robert P.
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 120 Drawing Page(s)
LN.CNT 6155
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Helicobacter ***pylori*** is known to cause or be a cofactor in type B ***gastritis*** , peptic ulcers, and ***gastric*** tumors. In both developed and developing countries, a high percentage of people are infected with this bacterium. The present invention relates generally to a certain H. ***pylori*** region located 5' to the CagA gene locus, to proteins encoded thereby, and to the use of these genes and proteins for diagnostic and vaccine applications.

L11 ANSWER 156 OF 184 USPATFULL
AN 1999:43190 USPATFULL
TI Method for stimulating production of variable region gene family restricted ***antibodies*** through B-cell superantigen vaccination
IN Silverman, Gregg J., Encinitas, CA, United States
PA The Regents of the University of California, Oakland, CA, United States (U.S. corporation)
PI US 5891438 19990406
WO 9409818 19940511
AI US 1995-428197 19950714 (8)
WO 1993-US10555 19931029
19950714 PCT 371 date
19950714 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1992-969936, filed on 30 Oct 1992, now abandoned
DT Utility
EXNAM Primary Examiner: Feisee, Lila; Assistant Examiner: Ungar, Susan

LREP Fish & Richardson P.C.
CLMN Number of Claims: 3
ECL Exemplary Claim: 1,3
DRWN 29 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 2731
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Criteria for identifying potential B cell superantigens are disclosed, together with a method for determining whether these candidate antigens have B cell superantigenic activity. Methods for constructing and using a vaccine including B cell superantigens are also disclosed. Identification is based on characterizing the structure of Ig binding sites which interact with the candidate antigen assessment of Ig V region diversity on binding of candidate and conventional antigens, confirmation of sAg activity in interactions between candidate antigens and whole cells, confirmation of whether the candidate antigen induces B cell mitogenesis, determination of the earliest point in B cell development where cellular co-factors are required for sAg activity and, for reference, determination of V region usage in responder populations. Once a B cell superantigen is characterized, it is purified and conjugated by chemical means to a polysaccharide or glycoprotein component from a microbial capsule, cell wall, envelope or other component preferably using components which stimulate production of ***antibodies*** with the same V region restriction as ***antibodies*** whose production is stimulated by the B cell superantigen.

L11 ANSWER 157 OF 184 USPATFULL
AN 1999:40212 USPATFULL
TI ATP-dependent RNA helicase protein
IN Bandman, Olga, Mountain View, CA, United States
Guegler, Karl J., Menlo Park, CA, United States
Corley, Neil C., Mountain View, CA, United States
Shah, Purvi, Sunnyvale, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 5888792 19990330
AI US 1997-892256 19970711 (8)
DT Utility
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Slobodyomsky, Elijobette
LREP Incyte Pharmaceuticals, Inc.
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 14 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 2270
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a human ATP-dependent RNA helicase (ADRH-1) and polynucleotides which identify and encode ADRH-1. The invention also provides expression vectors, host cells, agonists, ***antibodies*** and antagonists. The invention also provides methods for treating disorders associated with expression of ADRH-1.

L11 ANSWER 158 OF 184 USPATFULL
AN 1999:30582 USPATFULL
TI Methods and compositions for the diagnosis of extraesophageal

gastric reflux
IN Koufman, James, Winston-Salem, NC, United States
PA Wake Forest University, Winston-Salem, NC, United States (U.S. corporation)
PI US 5879897 19990309
AI US 1996-717793 19960923 (8)
DT Utility
EXNAM Primary Examiner: Saunders, David
LREP Arnold, White & Durkee
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1865
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are methods of detecting a ***gastric*** reflux in the esophagus or in the throat of a subject. The basis of the method is the detection of the presence of pepsin or ***pepsinogen*** at higher than normal levels. Detection is preferably by an ***immunoassay*** technique.

L11 ANSWER 159 OF 184 USPATFULL
AN 1999:27407 USPATFULL
TI Helicobacter TagA gene fusion protein
IN Cover, Timothy L., Nashville, TN, United States
Blaser, Martin J., Nashville, TN, United States
Kleanthous, Harry, Cambridge, MA, United States
Tummuru, Murali K. R., Nashville, TN, United States
PA Vanderbilt University, Nashville, TN, United States (U.S. corporation)
PI US 5876943 19990302
AI US 1998-34306 19980302
RLI Continuation of Ser. No. US 1994-316397, filed on 30 Sep 1994, now patented, Pat. No. US 5733340 which is a continuation-in-part of Ser. No. US 1993-53614, filed on 26 Apr 1993, now patented, Pat. No. US 5403924, issued on 4 Apr 1995 which is a continuation-in-part of Ser. No. US 1992-959940, filed on 13 Oct 1992, now abandoned
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Needle & Rosenberg
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 2470
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides an isolated nucleic acid encoding an approximately 120-128 kilodalton antigen of Helicobacter ***pylori*** , or an antigenic fragment thereof, wherein the antigen is associated with peptic ulceration. The present invention also provides methods of detecting the presence of a Helicobacter ***pylori*** strain possessing the 120-128 kilodalton antigen in a subject, comprising the steps of contacting an ***antibody*** -containing sample from the subject with a detectable amount of the tagA antigen or antigenic polypeptide of the present invention and detecting the binding of the antigen or fragment and the ***antibody*** . The detection of a strain expressing the TagA antigen is an indication of predisposition to

peptic ulceration and ***gastric*** carcinoma. A mutant H. ***pylori*** not expressing a functional TagA antigen is also provided.

L11 ANSWER 160 OF 184 USPATFULL
AN 1999:21925 USPATFULL
TI ***Immunoassay*** for H. ***pylori*** in fecal specimens
IN Larka, Christopher Vance, Cincinnati, OH, United States
Yi, Ching Sui Arthur, Cincinnati, OH, United States
Kozak, Kenneth James, Cincinnati, OH, United States
PA Meridian Diagnostics, Inc., Cincinnati, OH, United States (U.S. corporation)
PI US 5871942 19990216
AI US 1997-897732 19970721 (8)
RLI Continuation-in-part of Ser. No. US 1996-647115, filed on 9 May 1996, now patented, Pat. No. US 5716791
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Thompson Hine & Flory LLP
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 1 Drawing Page(s)
LN.CNT 502
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A process for the determination of H. ***pylori*** in a fecal specimen which comprises; (a) collecting a smear of a fecal specimen on a substrate; (b) immersing at least the portion of the substrate carrying the smear in a sample diluent so as to disperse the fecal specimen in the diluent; (c) contacting the fecal specimen in the diluent with a first polyclonal ***antibody*** for H. ***pylori*** antigen to form a complex of the ***antibody*** and the antigen; (d) separating the specimen and the complex; (e) exposing the complex to a second polyclonal ***antibody*** for the antigen and a portion of the ***antibody*** reacting with the complex, one of said first and second ***antibody*** being bound to a solid carrier and the other being labelled with a detection agent; and (f) determining the amount of the labelled ***antibody*** and in turn determining the presence of H. ***pylori*** antigen in the fecal specimen.

L11 ANSWER 161 OF 184 USPATFULL
AN 1999:4417 USPATFULL
TI Human lysophospholipase
IN Hillman, Jennifer L., Mountain View, CA, United States
Shah, Purvi, Sunnyvale, CA, United States
Murry, Lynn E., Portola Valley, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 5858756 19990112
AI US 1997-844120 19970429 (8)
DT Utility
EXNAM Primary Examiner: Hendricks, Keith D.; Assistant Examiner: Mayhew, Bradley S.
LREP Incyte Pharmaceuticals, Inc.; Billings, Lucy J.; Mohan-Peterson, Sheela
CLMN Number of Claims: 8

ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1990
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a human lysophospholipase (NHLP) and polynucleotides which identify and encode NHLP. The invention also provides expression vectors, host cells, agonists, ***antibodies*** and antagonists. In addition, the invention provides methods for producing NHLP and for treating or preventing disorders associated with expression of NHLP.

L11 ANSWER 162 OF 184 USPATFULL
AN 1998:154068 USPATFULL
TI Test kits and methods for detecting H. ***pylori***
IN Pronovost, Allan David, San Diego, CA, United States
Pawlak, Jan Waclaw, Cardiff, CA, United States
Condon, Kristy S., San Diego, CA, United States
PA Quidel Corporation, San Diego, CA, United States (U.S. corporation)
PI US 5846751 19981208
AI US 1995-486843 19950607 (8)
RLI Division of Ser. No. US 1994-292932, filed on 18 Aug 1994 which is a continuation of Ser. No. US 1993-22817, filed on 24 Feb 1993, now abandoned which is a continuation of Ser. No. US 1990-621845, filed on 4 Dec 1990, now abandoned
DT Utility
EXNAM Primary Examiner: Loring, Susan A.
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 1 Drawing Figure(s); 1 Drawing Page(s)
LN.CNT 725
AB A sensitive and specific antigen preparation for the detection of Helicobacter ***pylori*** in biological samples is disclosed. The preparation uses a range of antigens derived from size exclusion chromatography of detergent-solubilized H. ***pylori*** cells. Serological assays such as ELISA, latex agglutination, and rapid EIA assays utilizing the improved antigen preparation, and a kit for use in these serological assays are also disclosed.

L11 ANSWER 163 OF 184 USPATFULL
AN 1998:143906 USPATFULL
TI Helicobacter ***pylori*** haemagglutinin protease protein, nucleic acid encoding therefor and ***antibodies*** specific thereto
IN Smith, Andrew William, Kent, Great Britain
PA Reckitt & Colman Products Limited, London, United Kingdom (non-U.S. corporation)
PI US 5837502 19981117
WO 9501445 19950112
AI US 1996-578516 19960916 (8)
WO 1994-GB1406 19940629
19960916 PCT 371 date
19960916 PCT 102(e) date
PRAI GB 1993-13437 19930630
DT Utility
EXNAM Primary Examiner: Horlick, Kenneth R.; Assistant Examiner: Tung, Joyce
LREP Wolf, Greenfield & Sacks, P.C.

CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 714
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Helicobacter ***pylori*** (H. ***pylori***) haemagglutinin/protease protein, nucleic acids encoding therefor and ***antibodies*** specific thereto are described and, in particular, to their use in the identification of H. ***pylori*** and in the diagnosis of H. ***pylori*** infection. Also described are kits for the identification and diagnosis of H. ***pylori*** infection.

L11 ANSWER 164 OF 184 USPATFULL
AN 1998:122236 USPATFULL
TI DNA encoding a histamine H2 receptor
IN Murry, Lynn E., Portola Valley, CA, United States
Au-Young, Janice, Berkeley, CA, United States
Guegler, Karl J., Menlo Park, CA, United States
Goli, Surya K., Sunnyvale, CA, United States
PA Incyte Pharmaceuticals, Palo Alto, CA, United States (U.S. corporation)
PI US 5817480 19981006
AI US 1996-748485 19961107 (8)
DT Utility
EXNAM Primary Examiner: Walsh, Stephen; Assistant Examiner: Basham, Daryl A.
LREP Billings, Lucy J.
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN 14 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 2287
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a novel histamine H2 receptor (H2RH) and polynucleotides which identify and encode H2RH. The invention also provides genetically engineered expression vectors and host cells comprising the nucleic acid sequences encoding H2RH and a method for producing H2RH. The invention also provides for agonists, ***antibodies*** , or antagonists specifically binding H2RH, and their use, in the prevention and treatment of diseases in which H2RH is expressed. Additionally, the invention provides for the use of antisense molecules to polynucleotides encoding H2RH for the treatment of diseases associated with the expression of H2RH. The invention also provides diagnostic assays which utilize the polynucleotide, or fragments or the complement thereof, and ***antibodies*** specifically binding H2RH.

L11 ANSWER 165 OF 184 USPATFULL
AN 1998:118977 USPATFULL
TI Antigen preparation for detecting H. ***pylori***
IN Pronovost, Allan David, San Diego, CA, United States
Pawlak, Jan Waclaw, Cardiff, CA, United States
Condon, Kristy S., San Diego, CA, United States
PA Quidel Corporation, San Diego, CA, United States (U.S. corporation)
PI US 5814455 19980929
AI US 2929325 19940818 (8)
RLI Continuation of Ser. No. 22817, filed on 24 Feb 1993, now abandoned which is a continuation of Ser. No. 621845, filed on 4 Dec 1990, now abandoned

DT Utility
EXNAM Primary Examiner: Sidberry, Hazel F.
LREP Morrison & Foerster
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN 1 Drawing Figure(s); 1 Drawing Page(s)
LN.CNT 730
AB A sensitive and specific antigen preparation for the detection of Helicobacter ***pylori*** in biological samples is disclosed. The preparation uses a range of antigens derived from size exclusion chromatography of detergent-solubilized H. ***pylori*** cells. Serological assays such as ELISA, latex agglutination, and rapid EIA assays utilizing the improved antigen preparation, and a kit for use in these serological assays are also disclosed.

L11 ANSWER 166 OF 184 USPATFULL
AN 1998:111817 USPATFULL
TI GDP-L-fucose: .beta.-D-galactoside 2-.alpha.-L-fucosyltransferases, DNA sequences encoding the same, method for producing the same and a method of genotyping a person
IN Lowe, John B., 3125 Bolgos Cir., Ann Arbor, MI, United States 48105
Lennon, Gregory, 8309 Norris Canyon, Castro Valley, CA, United States 94552
Rouquier, Sylvie, 5, rue du Cannau, 34000 Montpellier, France
Giorgi, Dominique, 5, rue du Cannau, 34000 Montpellier, France
Kelly, Robert J., 3164 Concord, Trenton, MI, United States 48183
PI US 5807732 19980915
AI US 1995-395800 19950228 (8)
DT Utility
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Nashed, Nashaat T.
LREP Oblon, Spivak, McClelland, Maier & Neustadt, P.C.
CLMN Number of Claims: 12
ECL Exemplary Claim: 9
DRWN 30 Drawing Figure(s); 23 Drawing Page(s)
LN.CNT 2647
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The gene encoding GDP-L-fucose: .beta.-D-Galactoside 2-.alpha.-L-fucosyltransferase has been cloned, and a mutation in this gene has been found to be responsible for an individual being a non-secretor.

L11 ANSWER 167 OF 184 USPATFULL
AN 1998:88644 USPATFULL
TI F.sub.0 ATP synthase subunit
IN Hillman, Jennifer L., Mountain View, CA, United States
Goli, Surya K., Sunnyvale, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 5786150 19980728
AI US 1997-815177 19970311 (8)
DT Utility
EXNAM Primary Examiner: Patterson, Jr., Charles L.
LREP Billings, Lucy J.; Mohan-Peterson, SheelaIncyte Pharmceuticals
CLMN Number of Claims: 10
ECL Exemplary Claim: 1

DRWN 4 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 1940
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a human ATP synthase subunit (ASYS) and polynucleotides which encode ASYS. The invention also provides expression vectors, host cells, agonists, antisense molecules, ***antibodies*** , or antagonists. The invention also provides methods for producing ASYS and for treating disorders associated with expression of ASYS.

L11 ANSWER 168 OF 184 USPATFULL
AN 1998:82577 USPATFULL
TI IceA gene and related methods
IN Miller, Geraldine G., Franklin, TN, United States
Peek, Jr., Richard M., Nashville, TN, United States
Thompson, Stuart A., Whites Creek, TN, United States
Blaser, Martin J., Nashville, TN, United States
PA Vanderbilt University, Nashville, TN, United States (U.S. corporation)
PI US 5780278 19980714
AI US 1996-650528 19960520 (8)
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Needle & Rosenberg, P.C.
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 2020
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A purified IceA protein of Helicobacter ***pylori*** is provided. The protein is expressed as either an IceA 1 or an IceA 2 variant. A purified polypeptide fragment of the IceA protein is also provided. An antigenic fragment of IceA is provided. An isolated nucleic acid that encodes an IceA protein of H. ***pylori*** is provided. A nucleic acid that encodes an IceA 1 variant and a nucleic acid that encodes an IceA 2 variant is also provided. Fragments of the iceA gene are provided. A method of detecting the presence of an ***antibody*** against H. ***pylori*** in a sample is provided. The method comprises the following steps: a) contacting the sample with a purified IceA protein of H. ***pylori*** or a H. ***pylori*** -specific fragment thereof; and b) detecting the binding of the ***antibody*** in the sample to the protein or fragment, the detection of binding indicating the presence in the sample of ***antibodies*** against H. ***pylori*** . A method of detecting the presence of an ***antibody*** against an ulcerative Helicobacter ***pylori*** strain in a sample is also provided.

L11 ANSWER 169 OF 184 USPATFULL
AN 1998:72414 USPATFULL
TI Methods for the diagnosis of diabetes and prediabetic conditions
IN MacKay, Ian Reay, Malvern, Australia
Rowley, Merrill Joy, Camberwell, Australia
Zimmet, Paul Zev, Toorak, Australia
PA Monash University, Clayton, Australia (non-U.S. corporation)
PI US 5770381 19980623

WO 9418568 19940818
AI US 1995-495584 19951010 (8)
WO 1994-AU56 19940209
19951010 PCT 371 date
19951010 PCT 102(e) date
PRAI AU 1993-7168 19930209
DT Utility
EXNAM Primary Examiner: Hendricks, Keith D.
LREP Foley & Lardner
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 1065
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for detecting autoantibodies to glutamic acid decarboxylase (GAD) in the serum of a patient as diagnostic of a diabetic or prediabetic condition in the patient, comprises contacting a serum sample from the patient with a GAD antigen and detecting binding of autoantibodies to GAD in the sample by the GAD antigen, wherein the GAD antigen comprises a GAD preparation containing an enhanced amount of dimer(s) or oligomer(s) of the 65 kD or 67 kD isoforms, or both, of GAD. A diagnostic kit is also inclosed.

L11 ANSWER 170 OF 184 USPATFULL
AN 1998:65037 USPATFULL
TI CDNA encoding a human ATP synthase Fo subunit (ASYSD)
IN Hillman, Jennifer L., Mountain View, CA, United States
Goli, Surya K., Sunnyvale, CA, United States
PA Incyte Pharmaceuticals, Inc., Palo Alto, CA, United States (U.S. corporation)
PI US 5763248 19980609
AI US 1997-948195 19971009 (8)
RLI Continuation of Ser. No. US 1997-819395, filed on 17 Mar 1997, now abandoned
DT Utility
EXNAM Primary Examiner: LeGuyader, John L.; Assistant Examiner: Wang, Andrew
LREP Billings, Lucy J.
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1963
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a human ATP synthase d subunit (ASYSD) and polynucleotides which encode ASYSD. The invention also provides expression vectors, host cells, agonists, antisense molecules, ***antibodies*** , or antagonists. The invention also provides methods for producing ASYSD and for treating disorders associated with expression of ASYSD.

L11 ANSWER 171 OF 184 USPATFULL
AN 1998:33767 USPATFULL
TI Taga gene and methods for detecting predisposition to peptic ulceration and ***gastric*** carcinoma
IN Cover, Timothy L., Nashville, TN, United States
Blaser, Martin J., Nashville, TN, United States

Kleanthous, Harry, Cambridge, MA, United States
Tummuru, Murali K. R., Nashville, TN, United States
PA Vanderbilt University, Nashville, TN, United States (U.S. corporation)
OraVax, Inc., Cambridge, MA, United States (U.S. corporation)
PI US 5733740 19980331
AI US 1994-316397 19940930 (8)
RLI Continuation-in-part of Ser. No. US 1993-53614, filed on 26 Apr 1993, now patented, Pat. No. US 5403924 which is a continuation-in-part of Ser. No. US 1992-959940, filed on 13 Oct 1992, now abandoned
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Needle & Rosenberg, P.C.
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 2520
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An isolated nucleic acid encoding an approximately 120-128 kilodalton antigen of Helicobacter ***pylori*** , or an antigenic fragment thereof, wherein the antigen is associated with peptic ulceration. The present invention also provides methods of detecting the presence of a Helicobacter ***pylori*** strain possessing the 120-128 kilodalton antigen in a subject, comprising the steps of contacting an ***antibody*** -containing sample from the subject with a detectable amount of the tagA antigen or antigenic polypeptide of the present invention and detecting the binding of the antigen or fragment and the ***antibody*** . The detection of a strain expressing the TagA antigen is an indication of predisposition to peptic ulceration and ***gastric*** carcinoma. A mutant H. ***pylori*** not expressing a functional TagA antigen is also provided.

L11 ANSWER 172 OF 184 USPATFULL
AN 1998:14644 USPATFULL
TI ***Immunoassay*** for H. ***pylori*** in fecal specimens
IN Larka, Christopher Vance, Cincinnati, OH, United States
Yi, Ching Sui Arthur, Cincinnati, OH, United States
Kozak, Kenneth James, Cincinnati, OH, United States
PA Meridian Diagnostics, Inc., Cincinnati, OH, United States (U.S. corporation)
PI US 5716791 19980210
AI US 1996-647115 19960509 (8)
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Thompson Hine & Flory LLP
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 457
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A process for the determination of H. ***pylori*** in a fecal specimen comprising (a) dispersing a fecal specimen suspected of carrying H. ***pylori*** in a sample diluent; (b) contacting the fecal specimen in the diluent with a first polyclonal ***antibody***

for H. ***pylori*** antigen to form a complex of the ***antibody*** and the antigen; (c) separating said specimen and said complex; (d) exposing the complex to a second polyclonal ***antibody*** for said antigen and a portion of the ***antibody*** reacting with said complex, one of said first and second ***antibody*** being bound to a solid carrier and the other being labeled with a detection agent; and (e) determining the amount of the labeled ***antibody*** and in turn determining the presence of H. ***pylori*** antigen in said fecal specimen.

L11 ANSWER 173 OF 184 USPATFULL
AN 1998:11876 USPATFULL
TI Platinum-containing compounds, methods for their preparation and applications thereof
IN Houthoff, Hendrik J., Amsterdam, Netherlands
Reedijk, Jan, Leiden, Netherlands
Jelsma, Tinka, Almere, Netherlands
Van Es, Remco Maria, Koog a/d Zaan, Netherlands
van den Berg, Franciscus Michiel, Hoofddorp, Netherlands
Lempers, Edwin Leo Marlo, Julianadorp, Netherlands
Bloemink, Marieke Johanna, Oegstgeest, Netherlands
PA Kreatech Diagnostics, Amsterdam, Netherlands (non-U.S. corporation)
PI US 5714327 19980203
AI US 1995-470265 19950606 (8)
RLI Continuation-in-part of Ser. No. US 1993-975586, filed on 29 Oct 1993, now patented, Pat. No. US 5580990, issued on 3 Dec 1996
PRAI NL 1990-1639 19900719
DT Utility
EXNAM Primary Examiner: Guzo, David; Assistant Examiner: Sandals, William
LREP Hoffmann & Baron, LLP
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1543
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides platinum-based probe compounds having the structure: ##STR1## wherein: Pt is a platinum atom, PROBE is a probe biomolecule for associating to a target biomolecule, M is a detectable marker moiety, and X and Y are stabilizing substituents. Also provided are platinum-based labeling compounds having the structure: ##STR2## wherein: Pt is a platinum atom, M is a detectable marker moiety, A is a displaceable leaving group, and X and Y are stabilizing substituents. The invention further provides platinum-based linker compounds having the structure: ##STR3## wherein: Pt is a platinum atom, A and B are the same or different reactive moieties, and X and Y are stabilizing substituents. Other Pt.sup.II and Pt.sup.IV compounds are also provided. Moreover, the invention provides methods for the preparation and use of these compounds, as well as diagnostic kits which contain the compounds.

L11 ANSWER 174 OF 184 USPATFULL
AN 97:20430 USPATFULL
TI Isolated Helicobacter hepaticus
IN Ward, Jerrold M., Gaithersburg, MD, United States
Fox, James G., Harvard, MA, United States
Collins, Jr., Michael J., Laurel, MD, United States

Gorelick, Peter L., Frederick, MD, United States
Benveniste, Raoul E., Bethesda, MD, United States
Tully, Joseph G., Germantown, MD, United States
Gonda, Matthew A., Walkersville, MD, United States
Paster, Bruce J., Lee, NH, United States
Dewhirst, III, Floyd E., Medfield, MA, United States
PA The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)
PI US 5610060 19970311
AI US 1994-266414 19940624 (8)
DT Utility
EXNAM Primary Examiner: Rollins, John W.; Assistant Examiner: Ware, Deborah
LREP Needle & Rosenberg, P.C.
CLMN Number of Claims: 1
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1816
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An isolated bacterium of the genus Helicobacter, characterized by the 16S ribosomal RNA encoding nucleotide sequence defined in the Sequence Listing as SEQ ID NO:1 is provided. An isolated nucleic acid having the nucleotide sequence defined in the Sequence Listing as SEQ ID NO:1 is provided. Such a nucleic acid can be used for diagnosis of infection with H. hepaticus. A nucleic acid of the present invention in a vector suitable for expression of the nucleic acid is also provided. The vector can be in a host suitable for expressing the nucleic acid. A purified antigen specific for H. hepaticus is provided. A method of making an animal model for chronic Helicobacter infection is also provided.

L11 ANSWER 175 OF 184 USPATFULL
AN 96:96934 USPATFULL
TI Methods and compositions for the detection and treatment of diseases associated with antigens of microorganisms
IN Calenoff, Emanuel, Chicago, IL, United States
PA Enteron, L.P., Oak Brook, IL, United States (U.S. corporation)
PI US 5567594 19961022
AI US 1993-170017 19931220 (8)
RLI Continuation-in-part of Ser. No. US 1991-693232, filed on 26 Apr 1991, now abandoned
DT Utility
EXNAM Primary Examiner: Knode, Marian C.; Assistant Examiner: Duffy, Patricia A.
LREP Brinks Hofer Gilson & Lione
CLMN Number of Claims: 15
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1943
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A library of isolated and purified antigens specific for a microorganism is a set of individual molecules. The library forms antigen-***antibody*** complexes useful in the context of diagnosing and treating conditions associated with a specific microorganism such as H. ***pylori*** -induced gastro-duodenal disease. For the antigen-***antibody*** complexes in question the ***antibody*** is an immunoglobulin, which is IgE if the antigens are allergens. Complexes

with IgA, IgG and IgM are also useful. By this multivariate approach, a specific condition is diagnosed with high sensitivity and specificity by determining whether complexes form between a specific antigen library and a biological sample which contains immunoglobulins from an individual. Such libraries also are useful for immunotherapy.

L11 ANSWER 176 OF 184 USPATFULL
AN 96:91959 USPATFULL
TI TNF receptor-associated intracellular signaling proteins and methods of use
IN Goeddel, David V., South San Francisco, CA, United States
Hsu, Hailing, South San Francisco, CA, United States
PA Tularik, Inc., So. San Francisco, CA, United States (U.S. corporation)
PI US 5563039 19961008
AI US 1995-414625 19950331 (8)
DT Utility
EXNAM Primary Examiner: Ulm, John
LREP Flehr, Hohbach, Test, Albritton & Herbert
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1317
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A novel family of intracellular signaling proteins, exemplified by a Tumor Necrosis Factor Receptor-1 Associated Death Domain protein (TRADD), share a common TRADD sequence and include transducers of signals that modulate cell growth, differentiation and apoptosis. As such, the TRADD proteins, TRADD-encoding nucleic acids, and natural TRADD intracellular binding targets provide both important targets and means for therapeutic intervention. In particular, the invention provides isolated TRADDs and TRADD fragments, nucleic acids encoding the subject TRADDs and TRADD fragments or capable of selectively hybridizing to such TRADD-encoding nucleic acids, vectors and cells comprising TRADD-encoding nucleic acids, and TRADD-specific binding reagents. These compositions find use in diagnostic and therapeutic methods for disease associated with undesirable cell growth, migration, differentiation and/or cytokine signal responsiveness and methods and compositions for identifying lead compounds and pharmacological agents.

L11 ANSWER 177 OF 184 USPATFULL
AN 96:75290 USPATFULL
TI Helicobacter ***pylori*** bacterial derived factor
IN Anton, Peter A., West Hollywood, CA, United States
Reeve, Jr., Joseph R., Oakhurst, CA, United States
Walsh, John H., Los Angeles, CA, United States
Faull, Kym F., Los Angeles, CA, United States
PA The Regents of the University of California, Oakland, CA, United States (U.S. corporation)
PI US 5547844 19960820
AI US 1995-395495 19950223 (8)
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Portner, Ginny Allen
LREP Rowland, Bertram I.
CLMN Number of Claims: 7

ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 507
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Chemotactin, diethyl phthalate, is shown to be a chemoattractant secreted by H. ***pylori*** . Chemotactin attracts phagocytic cells with a resulting inflammatory episode. Chemotactin and its metabolites may be used for diagnosis and monitoring courses of infection by H. ***pylori*** or other chemotactin secreting organisms. In addition, chemotactin may be used in research for studying the inflammatory process, for identifying new drugs for modulating chemoattraction and activation of phagocytic cells, and for inducing an inflammatory response as a therapeutic intervention.

L11 ANSWER 178 OF 184 USPATFULL
AN 96:53195 USPATFULL
TI CagB and CagC genes of helicobacter ***pylori*** and related compositions
IN Blaser, Martin J., Nashville, TN, United States
Tummuru, Murali K. R., Nashville, TN, United States
Sharma, Smita A., Nashville, TN, United States
PA Vanderbilt University, Nashville, TN, United States (U.S. corporation)
PI US 5527678 19960618
AI US 1994-327494 19941021 (8)
DT Utility
EXNAM Primary Examiner: Jones, W. Gary; Assistant Examiner: Rees, Dianne
LREP Needle & Rosenberg
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1854
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A cagB gene of H. ***pylori*** is provided. This nucleic acid can be the nucleic acid consisting of nucleotides 193 through 1158 in the sequence set forth as SEQ ID NO:1, which is an example of a native coding sequence for CagB. This nucleic acid can also be in a vector suitable for expressing a polypeptide encoded by the nucleic acid. A cagC gene of H. ***pylori*** is provided. This nucleic acid can be the isolated nucleic acid consisting of nucleotides 1170 through 3830 in the sequence set forth as SEQ ID NO:3, which is an example of a native coding sequence for CagC. This nucleic acid can also be in a vector suitable for expressing a polypeptide encoded by the nucleic acid. Isolated nucleic acids that specifically hybridize with cagB and cagC are provided. CagB and CagC are associated with peptic ulceration and other clinical syndromes in humans infected with strains of H. ***pylori*** that express it.

L11 ANSWER 179 OF 184 USPATFULL
AN 95:101215 USPATFULL
TI Receptor conjugates for targeting penicillin antibiotics to bacteria
IN Krivan, Howard C., Derwood, MD, United States
Blomberg, A. Lennart I., Lund, Sweden
PA MicroCarb, Inc., Gaithersburg, MD, United States (U.S. corporation)
PI US 5466681 19951114
AI US 1994-180397 19940112 (8)

RLI Continuation of Ser. No. US 1990-484568, filed on 23 Feb 1990, now abandoned
DT Utility
EXNAM Primary Examiner: Walsh, Stephen G.; Assistant Examiner: Kemmerer, Elizabeth C.
LREP Pennie & Edmonds
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 719
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A variety of conjugates useful for the treatment of infections due to pathogenic microorganisms are provided. The conjugates comprise at least one agent coupled to a receptor which binds a microorganism. Suitable agents include anti-infectives, such as antibiotics and synthetic drugs. The present invention also provides methods for treating infections in warm-blooded animals due to pathogenic microorganisms.

L11 ANSWER 180 OF 184 USPATFULL
AN 95:92690 USPATFULL
TI Campylobacter ***pylori*** antigens and uses thereof for detection of Campylobacter ***pylori*** infection
IN Blaser, Martin J., New York, NY, United States
Perez-Perez, Guillermo I., Denver, CO, United States
PA Enteric Research Laboratories, Inc., Denver, CO, United States (U.S. corporation)
PI US 5459041 19951017
AI US 1988-158003 19880218 (7)
DT Utility
EXNAM Primary Examiner: Spiegel, Carol A.
LREP Ostrolenk, Faber, Gerb & Soffen
CLMN Number of Claims: 30
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 9 Drawing Page(s)
LN.CNT 1279
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Antigenic compositions are disclosed for use in diagnostic kits and the like for detecting the presence of ***antibodies*** specific for Campylobacter ***pylori*** , bacteria often associated with the occurrence of Type B ***gastritis*** and peptic ulcer disease. Samples of bodily fluids, for instance, may be contacted with immobilized antigen which is then washed and tested for the occurrence of significant levels of antigen/ ***antibody*** complex. Levels exceeding a predetermined positive threshold are indicative of ***antibodies*** to Campylobacter ***pylori*** in the sample tested. Kits employing the antigenic compositions of the invention preferably include means for detecting the antigen/ ***antibody*** complex such as materials and reagents for conducting an enzyme-linked immunosorbent assay, Western blot technique, liposome-based assay or other known detection tests.

L11 ANSWER 181 OF 184 USPATFULL
AN 95:47611 USPATFULL
TI Rapid in vitro test for helicobacter ***pylori*** using saliva
IN Cripps, Allan, East Maitland, Australia

Witt, Campbell, Bicton, Australia
Clancy, Robert L., New Lambton, Australia
Stiel, Daniel, East Lindfield, Australia
PA Auspharm International Ltd., New South Wales, Australia (non-U.S. corporation)
PI US 5420014 19950530
AI US 1992-876524 19920430 (7)
DT Utility
EXNAM Primary Examiner: Bidwell, Carol E.
LREP Scully, Scott, Murphy & Presser
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 638
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention contemplates a method for detecting contempory infection by H. ***pylori*** in a mammal comprising contacting a mucous secretion from said mammal with an antigen component from H. ***pylori*** for a time and under conditions sufficient for an IgG ***antibody*** in said mucous secretion specific to said antigen component to form a complex therewith and then subjecting said complex to a detecting means. Preferably, the antigen component is immobilized onto a solid support.

L11 ANSWER 182 OF 184 USPATFULL
AN 95:29722 USPATFULL
TI Taga gene and methods for detecting predisposition to peptic ulceration
IN Cover, Timothy L., Nashville, TN, United States
Tummuru, Murali K. R., Nashville, TN, United States
Blaser, Martin J., Nashville, TN, United States
PA Vanderbilt University, Nashville, TN, United States (U.S. corporation)
PI US 5403924 19950404
AI US 1993-53614 19930426 (8)
RLI Continuation-in-part of Ser. No. US 1992-959940, filed on 13 Oct 1992, now abandoned
DT Utility
EXNAM Primary Examiner: Parr, Margaret; Assistant Examiner: Campbell, Eggerton
LREP Needle & Rosenberg
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 1854
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides an isolated nucleic acid encoding an approximately 120-128 kilodalton antigen of Helicobacter ***pylori*** , or an antigenic fragment thereof, wherein the antigen is associated with peptic ulceration. The present invention also provides methods of detecting the presence of a Helicobacter ***pylori*** strain possessing the 120-128 kilodalton antigen in a subject, comprising the steps of contacting an ***antibody*** -containing sample from the subject with a detectable amount of the tagA antigen or antigenic fragment of the present invention and detecting the reaction of the antigen or fragment and the ***antibody*** . A mutant H. ***pylori*** not expressing a functional tagA antigen is also provided.

L11 ANSWER 183 OF 184 USPATFULL
AN 93:93550 USPATFULL
TI Method and product for the treatment of ***gastric*** disease
IN Cordle, Christopher T., Centerburg, OH, United States
Schaller, Joseph P., Columbus, OH, United States
PA Abbott Laboratories, Abbott Park, IL, United States (U.S. corporation)
PI US 5260057 19931109
AI US 1992-999233 19921231 (7)
RLI Division of Ser. No. US 1992-926181, filed on 7 Aug 1992 which is a continuation of Ser. No. US 1990-559793, filed on 30 Jul 1990, now abandoned
DT Utility
EXNAM Primary Examiner: Chan, Y. Christina; Assistant Examiner: Loring, Susan A.
LREP Drayer, Lonnie R.; Nickey, Donald O.
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 672
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention describes a product obtained from the isolation and concentration of specific immunoglobulins (***antibodies***) derived from the mammary secretions of cows immunized with Helicobacter ***pylori*** . The product is useful in preparing formulations for the treatment and/or prevention of ***gastric*** diseases.

L11 ANSWER 184 OF 184 USPATFULL
AN 93:91432 USPATFULL
TI Method and product for the treatment of ***gastric*** disease
IN Cordle, Christopher T., Centerburg, OH, United States
Schaller, Joseph P., Columbus, OH, United States
PA Abbott Laboratories, Abbott Park, IL, United States (U.S. corporation)
PI US 5258178 19931102
AI US 1992-926181 19920807 (7)
RLI Continuation of Ser. No. US 1990-559793, filed on 30 Jul 1990, now abandoned
DT Utility
EXNAM Primary Examiner: Lacey, David L.; Assistant Examiner: Futrovsky, Susan L.
LREP Drayer, Lonnie R.; Nickey, Donald O.
CLMN Number of Claims: 3
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 684
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention describes a product obtained from the isolation and concentration of specific immunoglobulins (***antibodies***) derived from the mammary secretions of cows immunized with Helicobacter ***pylori*** . The product is useful in preparing formulations for the treatment and/or prevention of ***gastric*** diseases.

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*File 155: First Medline 2001 update is expected towards the end of
February. For other NLM information see Help News155.
  File   5:Biosis Previews(R)  1969-2001/Feb W3
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  File  73:EMBASE  1974-2001/Feb W3
        (c) 2001 Elsevier Science B.V.
*File  73: For information about Explode feature please
see Help News73.
  File  94:JICST-EPlus  1985-2001/Feb W2
        (c)2001 Japan Science and Tech Corp(JST)
*File  94: There is no data missing. UDs have been adjusted to reflect
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09774848   99070725
   Serum antibodies to H+,K+- ATPase , serum pepsinogen A and
 Helicobacter pylori in relation to gastric mucosa morphology in
patients with low or low-normal concentrations of serum cobalamins.
Jul 1998
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6/6/2 (Item 2 from file: 155)
06651765 91047803
Acid and barriers. Current research and future developments for peptic ulcer therapy.
1990

6/6/3 (Item 1 from file: 5)
11244184 BIOSIS NO.: 199800025516
Helicobacter pylori **associated autoantibodies recognize Lewis antigens, and peptide epitopes of gastric H+, K+-** ATPase **and intrinsic factor.**
1997

6/6/4 (Item 1 from file: 94)
04051065 JICST ACCESSION NUMBER: 99A0425613 FILE SEGMENT: JICST-E
Physiology and pathology. 5. Gastric secretion mechanism., 1999
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ialog level 00.12.12D

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*** NEW Current Year Ranges Install ***

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(c) format only 2000 Dialog Corporation
*File 155: First Medline 2001 update is expected towards the end of February. For other NLM information see Help News155.
File 5:Biosis Previews(R) 1969-2001/Feb W3
(c) 2001 BIOSIS
File 73:EMBASE 1974-2001/Feb W3
(c) 2001 Elsevier Science B.V.
*File 73: For information about Explode feature please see Help News73.
File 94:JICST-EPlus 1985-2001/Feb W2
(c)2001 Japan Science and Tech Corp(JST)
*File 94: There is no data missing. UDs have been adjusted to reflect the current months data. See Help News94 for details.

Set	Items	Description

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6/9/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

06651765 91047803
Acid and barriers. Current research and future developments for peptic ulcer therapy.
Rademaker JW; Hunt RH
Division of Gastroenterology, McMaster University Medical Centre, Hamilton, Ontario, Canada.
Scandinavian journal of gastroenterology. Supplement (NORWAY) 1990, 175 p19-26, ISSN 0085-5928 Journal Code: UCT
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
JOURNAL ANNOUNCEMENT: 9102
Subfile: INDEX MEDICUS
Medical therapy for peptic ulcer disease has been targeted at inhibiting acid secretion based on the belief that ulcers occur due to an imbalance between aggressive and protective factors. New antisecretory agents are unlikely to show any dramatic improvement over the success and safety of histamine H2 receptor antagonists or the recently introduced H+K+**ATPase** proton pump antagonist omeprazole. The development of specific muscarinic M3 and gastrin receptor antagonists will provide useful agents to suppress acid and **pepsinogen** secretion by alternative means and may prevent the associated hypergastrinaemia seen with anti-secretory therapy. Enhancement of mucosal defence by site protective agents will be based on a better understanding of the vascular and immune factors involved in maintaining mucosal integrity and the growth factors that regulate wound healing. Molecular techniques are likely to produce the 'model anti-ulcer' agent which will effectively inhibit acid secretion and also enhance wound healing thus providing a cure for this chronic disease. (67 Refs.)
Tags: Human
Descriptors: *Antacids--Therapeutic Use--TU; *Anti-Ulcer Agents --Therapeutic Use--TU; *Peptic Ulcer--Drug Therapy--DT; Gastric Mucosa --Physiology--PH; **Helicobacter pylori** ; **Helicobacter** Infections --Complications--CO; Histamine H2 Antagonists--Therapeutic Use--TU; Intestinal Mucosa--Physiology--PH; Peptic Ulcer--Etiology--ET; Wound Healing--Physiology--PH
CAS Registry No.: 0 (Antacids); 0 (Anti-Ulcer Agents); 0 (Histamine H2 Antagonists)

6/9/3 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11244184 BIOSIS NO.: 199800025516
Helicobacter pylori **associated autoantibodies recognize Lewis antigens, and peptide epitopes of gastric H+, K+- ATPase and intrinsic factor.**
AUTHOR: Appelmelk B J(a); Straver S(a); Claeys D; Faller G; Kirchner T; Negrini R; Krakowka S; Eaton K; Vandenbroucke-Grauls C M J E(a)
AUTHOR ADDRESS: (a)Vrije Univ., Amsterdam**Netherlands
JOURNAL: Gut 41 (SUPPL. 1):pA17 1997
CONFERENCE/MEETING: European Helicobacter Pylori Study Group Xth International Workshop on Gastroduodenal Pathology and Helicobacter Pylori Lisbon, Portugal September 11-14, 1997
SPONSOR: European Helicobacter pylori Study Group
ISSN: 0017-5749
RECORD TYPE: Citation
LANGUAGE: English
REGISTRY NUMBERS: 9000-83-3: **ATPASE** ; 9001-10-9: **PEPSINOGEN**
DESCRIPTORS:
MAJOR CONCEPTS: Clinical Immunology (Human Medicine, Medical Sciences); Gastroenterology (Human Medicine, Medical Sciences); Infection
BIOSYSTEMATIC NAMES: Aerobic Helical or Vibrioid Gram-Negatives--Eubacteria, Bacteria, Microorganisms; Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia; Suidae--Artiodactyla, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: human (Hominidae)--patient; pig (Suidae); **Helicobacter -pylori** (Aerobic Helical or Vibrioid Gram-Negatives)
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Artiodactyls; Bacteria; Chordates; Eubacteria; Humans; Mammals; Microorganisms; Nonhuman Mammals; Nonhuman Vertebrates; Primates; Vertebrates
DISEASES: gastric atrophy--digestive system disease; type B gastritis--digestive system disease; **Helicobacter -pylori** infection--bacterial disease, digestive system disease, pathophysiology, pathogenesis
CHEMICALS & BIOCHEMICALS: gastric intrinsic factor; gastric proton, potassium ion-**ATPase** --peptide epitopes; **pepsinogen** ; **Helicobacter -pylori** -associated autoantibodies--Lewis antigen recognition
MISCELLANEOUS TERMS: Meeting Abstract
CONCEPT CODES:
34504 Immunology and Immunochemistry-Bacterial, Viral and Fungal
02508 Cytology and Cytochemistry-Human
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
10068 Biochemical Studies-Carbohydrates
10506 Biophysics-Molecular Properties and Macromolecules
10508 Biophysics-Membrane Phenomena
10806 Enzymes-Chemical and Physical
10808 Enzymes-Physiological Studies
14006 Digestive System-Pathology
15002 Blood, Blood-Forming Organs and Body Fluids-Blood and Lymph Studies
34506 Immunology and Immunochemistry-Immunohematology, Blood Groups
34508 Immunology and Immunochemistry-Immunopathology, Tissue Immunology
36002 Medical and Clinical Microbiology-Bacteriology
00520 General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
BIOSYSTEMATIC CODES:
06210 Aerobic Helical or Vibrioid Gram-Negatives (1992-)
85740 Suidae
86215 Hominidae

6/9/4 (Item 1 from file: 94)
DIALOG(R)File 94:JICST-EPlus

04051065 JICST ACCESSION NUMBER: 99A0425613 FILE SEGMENT: JICST-E
Physiology and pathology. 5. Gastric secretion mechanism.

NAKAMURA MASAHIKO (1); KISHIKAWA HIROSHI (2); ISHII HIROMASA (2)
(1) Tokyo Denryoku Hosp.; (2) Keio Univ.
Annual Review Shokaki, 1999, VOL.1999, PAGE.82-85, REF.21
JOURNAL NUMBER: L1627AAR
UNIVERSAL DECIMAL CLASSIFICATION: 616.3-09 591.132.2.05+591.433
LANGUAGE: Japanese COUNTRY OF PUBLICATION: Japan
DOCUMENT TYPE: Review
ARTICLE TYPE: Review article
MEDIA TYPE: Printed Publication
DESCRIPTORS: gastric juice secretion; smooth muscle cell; **ATPase** ; cotransport; ion transport; nitrogen monoxide; endothelin; **Helicobacter pylori** ; growth factor; parietal cell; **pepsinogen** ; gastric mucosa; fundus ventriculi; defence mechanism
BROADER DESCRIPTORS: external secretion; secretion(physiology); digestive system physiology; cell(cytology); nuclease; hydrolase; enzyme; pyrophosphatase; biological transport; transportation; nitrogen oxide; oxide; chalcogenide; oxygen group element compound; oxygen compound; nitrogen compound; nitrogen group element compound; bioactive peptide; peptide; **Helicobacter** ; spiral and curved bacteria; bacterium; microorganism; bioactive factor; factor; zymogen; precursor(substance); stomach; gastrointestinal duct; digestive organ; mucosa; epithelial tissue; animal tissue; biomedical tissue; organization; histomembrane; membrane and film; mechanism
CLASSIFICATION CODE(S): GH01020E; EJ06030C

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*** DIALINDEX search results display in an abbreviated ***
*** format unless you enter the SET DETAIL ON command. ***
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           2731     5: Biosis Previews(R)_1969-2001/Feb W3
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              2     8: Ei Compendex(R)_1970-2001/Feb W1
              3     9: Business & Industry(R)_Jul/1994-2001/Feb 22
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              1    15: ABI/Inform(R)_1971-2001/Feb 22
             12    16: Gale Group PROMT(R)_1990-2001/Feb 22
              4    19: CHEM.INDUSTRY NOTES_1974-2001/ISS 200108
              3    20: World Reporter_1997-2001/Feb 23
              5    28: Oceanic Abst._1964-2001/Mar
           1517    34: SciSearch(R) Cited Ref Sci_1990-2001/Feb W4
             52    35: Dissertation Abstracts Online_1861-2000/Dec
              3    41: Pollution Abs_1970-2001/Mar
              3    42: PHARMACEUTICAL NEWS INDEX_1974-2001/Feb W1
             29    44: Aquatic Sci&Fish Abs_1978-2001/Feb
             16    47: Gale Group Magazine DB(TM)_1959-2001/Feb 22
              2    48: SPORTDiscus_1962-2001/Feb
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             11    51: Food Sci.&Tech.Abs_1969-2001/Apr W4
              6    53: FOODLINE(R): Food Science &
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             72    65: Inside Conferences_1993-2001/Feb W3
              1    68: ENV.BIB._1974-2000/NOV
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           1653    73: EMBASE_1974-2001/Feb W3
              7    74: Int.Pharm.Abs._1970-2001/Jan
            248    76: Life Sciences Collection_1982-2001/Dec
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              1    79: Foods Adlibra(TM)_1974-2001/Feb
              4    91: MANTIS(TM)_1880-2000/Apr
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             31    98: General Sci Abs/Full-Text_1984-2001/Jan
             34   103: Energy SciTec_1974-2001/Feb B1
              6   107: Adis R&D Insight_1986-2001/Feb W4
              6   108: AEROSPACE DBASE_1962-2001/FEB
              4   109: Nuclear Sci. Abs._1948-1976
              2   124: CLAIMS(R)/REFERENCE_2000/Q3
              2   128: PHARMAPROJECTS_1980-2001/Feb W3
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            409   151: HealthSTAR_1975-2000/Dec
           2281   155: MEDLINE(R)_1966-2000/Dec W4
            335   156: Toxline(R)_1965-2000/Dec
             14   161: Occ.Saf.& Hth._1973-1998/Q3
            117   162: CAB HEALTH_1983-2001/Jan
             11   172: EMBASE Alert_2001/Feb W3
             10   174: Pharm-line(R)_1978-2001/Feb W1
              4   180: Federal Register_1985-2001/Feb 22
             29   185: Zoological Record Online(R)_1978-2001/Jan
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                2   187: F-D-C Reports_1987-2001/Feb W3
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                2   229: Drug Info._2000/Q3
               10   266: FEDRIP_2001/Feb
               14   285: BioBusiness(R)_1985-1998/Aug W1
                6   286: Biocommerce Abs.& Dir._1981-2001/Feb B1
                3   292: GEOBASE(TM)_1980-2001/Feb
                2   303: Chapman & Hall Chemical Database_1997/Apr
               23   305: Analytical Abstracts_1980-2001/Feb W2
                2   315: ChemEng & Biotec Abs_1970-2000/Dec
                8   319: Chem Bus NewsBase_1984-2001/Feb 23
                2   322: Polymer Online_
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               10   340: CLAIMS(R)/US PATENT_1950-01/Feb 20
                6   342: Derwent Patents Citation Indx_1978-00/200107
               11   345: Inpadoc/Fam.& Legal Stat_1968-2001/UD=200107
                8   347: JAPIO Oct 1976-2000/Jul(UPDATED 001114)
               26   348: EUROPEAN PATENTS_1978-2000/Feb W02
               79   349: PCT Fulltext_1983-2001/UB=20010215, UT=20010201
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                1   358: Current BioTech Abs_1983-1999/Dec
                1   371: French Patents_1961-2000/BOPI 0052
                4   390: Beilstein Online_
               95   398: CHEMSEARCH(TM)_1957-2001/Jan
             1139   399: CA SEARCH(R)_1967-2001/UD=13409
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               20   444: New England Journal of Med._1985-2001/Feb W4
                3   445: IMSWorld R&D Focus_1991-2001/Jan W4
                7   449: IMSWorld Company Profiles_1992-2001/Jan
               28   457: The Lancet_1986-2000/Oct W1
                2   461: USP DI(R) Vol. I_1998/Q3
                2   467: ExtraMED(tm)_2000/Dec
               32   484: Periodical Abstracts Plustext_1986-2001/Feb W3
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                1   613: PR Newswire_1999-2001/Feb 23
                1   621: Gale Group New Prod.Annou.(R)_1985-2001/Feb 22
                2   624: McGraw-Hill Publications_1985-2001/Feb 21
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                2   649: Gale Group Newswire ASAP(TM)_2001/Feb 20
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N7           903   144: Pascal_1973-2001/Feb W2
N8           865   434: SciSearch(R) Cited Ref Sci_1974-1989/Dec
N9           777    94: JICST-EPlus_1985-2001/Feb W2
N10          636    50: CAB Abstracts_1972-2001/Jan
   102 files have one or more items; file list includes 285 files.

       - Enter P or PAGE for more -
?p
Your last SELECT statement was:
   S PEPSINOGEN?

Ref         Items   File
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N11           409   151: HealthSTAR_1975-2000/Dec
N12           335   156: Toxline(R)_1965-2000/Dec
N13           315    71: ELSEVIER BIOBASE_1994-2001/Feb W4
N14           248    76: Life Sciences Collection_1982-2001/Dec
N15           227    10: AGRICOLA_70-2001/Feb
N16           117   162: CAB HEALTH_1983-2001/Jan
N17           110   203: AGRIS_1974-2001/Oct
N18            95   398: CHEMSEARCH(TM)_1957-2001/Jan
N19            79   349: PCT Fulltext_1983-2001/UB=20010215, UT=20010201
N20            78   654: US Pat.Full._1990-2001/Feb 20
   102 files have one or more items; file list includes 285 files.

       - Enter P or PAGE for more -
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Your last SELECT statement was:
   S PEPSINOGEN?

Ref         Items   File
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N21            72    65: Inside Conferences_1993-2001/Feb W3
N22            72    77: Conference Papers Index_1973-2001/Jan
N23            68   143: Biol. & Agric. Index_1983-2001/Jan
N24            63   149: TGG Health&Wellness DB(SM)_1976-2001/Feb W2
N25            52    35: Dissertation Abstracts Online_1861-2000/Dec
N26            34   103: Energy SciTec_1974-2001/Feb B1
N27            32   442: AMA  Journals_1982-2000/Oct B3
N28            32   484: Periodical Abstracts Plustext_1986-2001/Feb W3
N29            31    98: General Sci Abs/Full-Text_1984-2001/Jan
N30            29    44: Aquatic Sci&Fish Abs_1978-2001/Feb
   102 files have one or more items; file list includes 285 files.

       - Enter P or PAGE for more -
?p
Your last SELECT statement was:
   S PEPSINOGEN?

Ref         Items   File
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N31            29   148: Gale Group Trade & Industry DB_1976-2001/Feb 21
N32            29   185: Zoological Record Online(R)_1978-2001/Jan
N33            29   653: US Pat.Fulltext_1980-1989
N34            28   457: The Lancet_1986-2000/Oct W1
N35            26   348: EUROPEAN PATENTS_1978-2000/Feb W02
N36            26   357: Derwent Biotechnology Abs_1982-2001/Mar B1
N37            23   305: Analytical Abstracts_1980-2001/Feb W2
N38            20   444: New England Journal of Med._1985-2001/Feb W4
N39            16    47: Gale Group Magazine DB(TM)_1959-2001/Feb 22
N40            14   161: Occ.Saf.& Hth._1973-1998/Q3
   102 files have one or more items; file list includes 285 files.

       - Enter P or PAGE for more -
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?p
Your last SELECT statement was:
   S PEPSINOGEN?

Ref        Items   File
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N41           14   285: BioBusiness(R)_1985-1998/Aug W1
N42           12    16: Gale Group PROMT(R)_1990-2001/Feb 22
N43           11    51: Food Sci.&Tech.Abs_1969-2001/Apr W4
N44           11   172: EMBASE Alert_2001/Feb W3
N45           11   345: Inpadoc/Fam.& Legal Stat_1968-2001/UD=200107
N46           11   636: Gale Group Newsletter DB(TM)_1987-2001/Feb 22
N47           10   174: Pharm-line(R)_1978-2001/Feb W1
N48           10   266: FEDRIP_2001/Feb
N49           10   340: CLAIMS(R)/US PATENT_1950-01/Feb 20
N50            8   319: Chem Bus NewsBase_1984-2001/Feb 23
   102 files have one or more items; file list includes 285 files.

        - Enter P or PAGE for more -
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Your last SELECT statement was:
   S PEPSINOGEN?

Ref        Items   File
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N51            8   347: JAPIO_Oct 1976-2000/Jul(UPDATED 001114)
N52            7     6: NTIS_1964-2001/Mar W1
N53            7    74: Int.Pharm.Abs._1970-2001/Jan
N54            7   449: IMSWorld Company Profiles_1992-2001/Jan
N55            6    53: FOODLINE(R): Food Science & Technology_1972-2001/F
N56            6    70: SEDBASE_1996/Jan Q1
N57            6   107: Adis R&D Insight_1986-2001/Feb W4
N58            6   108: AEROSPACE DBASE_1962-2001/FEB
N59            6   286: Biocommerce Abs.& Dir._1981-2001/Feb B1
N60            6   342: Derwent Patents Citation Indx_1978-00/200107
   102 files have one or more items; file list includes 285 files.

        - Enter P or PAGE for more -
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Your last SELECT statement was:
   S PEPSINOGEN?

Ref        Items   File
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N61            6   764: BCC Market Research_1989-2001/Jan
N62            5     2: INSPEC_1969-2001/Feb W3
N63            5    28: Oceanic Abst._1964-2001/Mar
N64            4    19: CHEM.INDUSTRY NOTES_1974-2001/ISS 200108
N65            4    91: MANTIS(TM)_1880-2000/Apr
N66            4   109: Nuclear Sci. Abs._1948-1976
N67            4   180: Federal Register_1985-2001/Feb 22
N68            4   390: Beilstein Online_
N69            4   429: Adis Newsletters(Archive)_1982-2001/Dec 12
N70            3     9: Business & Industry(R)_Jul/1994-2001/Feb 22
   102 files have one or more items; file list includes 285 files.

        - Enter P or PAGE for more -
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Your last SELECT statement was:
   S PEPSINOGEN?

Ref        Items   File
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N71            3    20: World Reporter_1997-2001/Feb 23
N72            3    41: Pollution Abs_1970-2001/Mar
N73            3    42: PHARMACEUTICAL NEWS INDEX_1974-2001/Feb W1
N74            3   292: GEOBASE(TM)_1980-2001/Feb
```

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N75             3   445: IMSWorld R&D Focus_1991-2001/Jan W4
N76             3   652: US Patents Fulltext_1971-1979
N77             3   761: Datamonitor Market Res._1992-2000/Dec
N78             2    84: Ei Compendex(R)_1970-2001/Feb W1
N79             2    48: SPORTDiscus_1962-2001/Feb
N80             2   124: CLAIMS(R)/REFERENCE_2000/Q3
   102 files have one or more items; file list includes 285 files.

       - Enter P or PAGE for more -
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Your last SELECT statement was:
   S PEPSINOGEN?

Ref         Items   File
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N81             2   128: PHARMAPROJECTS_1980-2001/Feb W3
N82             2   187: F-D-C Reports_1987-2001/Feb W3
N83             2   229: Drug Info._2000/Q3
N84             2   303: Chapman & Hall Chemical Database_1997/Apr
N85             2   315: ChemEng & Biotec Abs_1970-2000/Dec
N86             2   322: Polymer Online_
N87             2   461: USP DI(R) Vol. I_1998/Q3
N88             2   467: ExtraMED(tm)_2000/Dec
N89             2   624: McGraw-Hill Publications_1985-2001/Feb 21
N90             2   649: Gale Group Newswire ASAP(TM)_2001/Feb 20
   102 files have one or more items; file list includes 285 files.

       - Enter P or PAGE for more -
?p
Your last SELECT statement was:
   S PEPSINOGEN?

Ref         Items   File
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N91             2   765: Frost & Sullivan_1992-1999/Apr
N92             1    15: ABI/Inform(R)_1971-2001/Feb 22
N93             1    68: ENV.BIB._1974-2000/NOV
N94             1    79: Foods Adlibra(TM)_1974-2001/Feb
N95             1   129: PHIND(Archival)_1980-2001/Feb W3
N96             1   358: Current BioTech Abs_1983-1999/Dec
N97             1   371: French Patents_1961-2000/BOPI 0052
N98             1   441: ESPICOM Pharm&Med DEVICE NEWS_2001/Feb W2
N99             1   583: Gale Group Globalbase(TM)_1986-2001/Feb 23
N100            1   613: PR Newswire_1999-2001/Feb 23
   102 files have one or more items; file list includes 285 files.

       - Enter P or PAGE for more -
?p
Your last SELECT statement was:
   S PEPSINOGEN?

Ref         Items   File
---         -----   ----
N101            1   621: Gale Group New Prod.Annou.(R)_1985-2001/Feb 22
N102            1   763: Freedonia Market Res._1990-2001/Feb
N103            0    14: Mechanical Engineering Abs_1973-2001/Mar
N104            0    18: Gale Group F&S Index(R)_1988-2001/Feb 22
N105            0    29: Meteor.& Geoastro.Abs._1970-2001/Mar
N106            0    31: World Surface Coatings Abs_1976-2001/Feb
N107            0    32: METADEX(R)_1966-2001/Apr B2
N108            0    33: Aluminium Ind Abs_1968-2001/Mar
N109            0    40: Enviroline(R)_1975-2001/Feb
N110            0    43: Health News Daily_1990-2001/Feb 14
   102 files have one or more items; file list includes 285 files.

       - Enter P or PAGE for more -
?b n2 n1 n3 n9;exs
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File 411:DIALINDEX(R)

DIALINDEX(R)
(c) 2001 The Dialog Corporation plc

*** DIALINDEX search results display in an abbreviated ***
*** format unless you enter the SET DETAIL ON command. ***
?sf biotech
You have 23 files in your file list.
(To see banners, use SHOW FILES command)
?show files

File	Name
5:	Biosis Previews(R)_1969-2001/Feb W1
6:	NTIS_1964-2001/Feb W4
8:	Ei Compendex(R)_1970-2001/Jan W2
34:	SciSearch(R) Cited Ref Sci_1990-2001/Feb W2
65:	Inside Conferences_1993-2001/Feb W2
71:	ELSEVIER BIOBASE_1994-2001/Jan W4
73:	EMBASE_1974-2001/Feb W1
76:	Life Sciences Collection_1982-2001/Dec
94:	JICST-EPlus_1985-2001/Jan W4
98:	General Sci Abs/Full-Text_1984-2001/Dec
99:	Wilson Appl. Sci & Tech Abs_1983-2001/Dec
143:	Biol. & Agric. Index_1983-2001/Dec
144:	Pascal_1973-2001/Feb W1
155:	MEDLINE(R)_1966-2000/Dec W4
172:	EMBASE Alert_2001/Feb W1
266:	FEDRIP_2001/Jan
315:	ChemEng & Biotec Abs_1970-2000/Dec
357:	Derwent Biotechnology Abs_1982-2001/Apr B1
358:	Current BioTech Abs_1983-1999/Dec
369:	New Scientist_1994-2001/Jan W4
370:	Science_1996-1999/Jul W3
399:	CA SEARCH(R)_1967-2001/UD=13407
434:	SciSearch(R) Cited Ref Sci_1974-1989/Dec

TD-001

Miki K, Fuchi et al. Serum pepsinogens as screening test of extensive chronic gastritis. Gastroenterol Jpn 1987; 22: 131-41

Furuta, T et al Journal of Clinical Gastroenterology 1995 Vol. 20 (Suppl 2) S107-S111

5/K/40 (Item 40 from file: 73)
DIALOG(R)File 73:EMBASE

06376091 EMBASE No: 1996040882
Serum pepsinogen I in childhood Helicobacter pylori gastritis: Its relation to mucosal peptic activity
Yahav J.; Oderda G.; Diver-Haber A.; Fradkin A.; Keller N.; Altare F.; Ansaldi N.; Jonas A.
Pediatric Gastroenterology Unit, Chaim Sheba Medical Center,52621 Tel-Hashomer Israel
Israel Journal of Medical Sciences (ISR. J. MED. SCI.) (Israel) 1996, 32/1 (56-59)
CODEN: IJMDA ISSN: 0021-2180
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

DRUG DESCRIPTORS:
***pepsinogen i** --endogenous compound--ec
MEDICAL DESCRIPTORS:
...adult; article; child; childhood disease--etiology--et; controlled study ; enzyme activity; enzyme blood level; female; **helicobacter pylori** ; human ; human tissue; major clinical study; male; pepsin secretion; stomach antrum
CAS REGISTRY NO.: 92228-49-4 (**pepsinogen i**); 9001-75-6 (pepsin a)
?
PLEASE ENTER A COMMAND OR BE LOGGED OFF IN 5 MINUTES
? t/s5/medium,k/46,49,60,64,65,66,69,70,71
>>>'5' valid only in keyword format
?t s5/medium,k/46,49,60,64,65,66,69,70,71

5/K/46 (Item 46 from file: 73)
DIALOG(R)File 73:EMBASE

06161628 EMBASE No: 1995161422
Decrease in basal serum pepsinogen I levels after H. pylori eradication
DESCENSO DE LOS NIVELES BASALES DE PEPSINOGENO I SERICO TRAS LA ERRADICACION DE H. PYLORI
Boixeda D.; Gisbert J.P.; Vila T.; Canton R.; Redondo C.; Martin De Argila C.; Cano A.; Garcia Plaza A.
C/Lagasca 13,28001 Madrid Spain
Revista Clinica Espanola (REV. CLIN. ESP.) (Spain) 1995, 195/4 (214-219)
CODEN: RCESA ISSN: 0014-2565
DOCUMENT TYPE: Journal; Article
LANGUAGE: SPANISH SUMMARY LANGUAGE: SPANISH; ENGLISH

DRUG DESCRIPTORS:
...*trial--ct; *clavulanic acid--drug combination--cb; *omeprazole--drug combination--cb; *omeprazole--clinical trial--ct; ***pepsinogen i** --endogenous compound--ec; *ranitidine--drug therapy--dt; *ranitidine--drug combination--cb; *ranitidine--clinical trial...
MEDICAL DESCRIPTORS:
***helicobacter pylori** ; *duodenum ulcer--drug therapy--dt; *gastritis--drug therapy--dt; *gram negative infection--drug therapy...
...CAS REGISTRY NO.: 95510-70-6 (omeprazole); 92228-49-4 (**pepsinogen i**); 66357-35-5...

5/K/49 (Item 49 from file: 73)
DIALOG(R)File 73:EMBASE

06083548 EMBASE No: 1995114035
Diagnosis of gastric adenocarcinoma using a scoring system: Combined

assay of serological markers of Helicobacter pylori infection, pepsinogen I and gastrin
Lin J.-T.; Lee W.-C.; Wu M.-S.; Wang J.-T.; Wang T.-H.; Chen C.-J.
Department of Internal Medicine, National Taiwan University Hospital, No 7 Chung-Shan S Rd, Sec 1,Taipei 10017 Taiwan
Journal of Gastroenterology (J. GASTROENTEROL.) (Japan) 1995, 30/2 (156-161)
CODEN: JOGAE ISSN: 0944-1174
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

DRUG DESCRIPTORS:
*gastrin--endogenous compound--ec; *pepsinogen i --endogenous compound--ec
MEDICAL DESCRIPTORS:
*helicobacter pylori ; *stomach adenocarcinoma--diagnosis--di
CAS REGISTRY NO.: 9002-76-0 (gastrin); 92228-49-4 (pepsinogen i)

5/K/60 (Item 60 from file: 73)
DIALOG(R)File 73:EMBASE

05042431 EMBASE No: 1992182647
Twenty-four-hour hyperpepsinogenaemia in Helicobacter pylori-positive subjects is abolished by eradication of the infection
Fraser A.G.; Prewett E.J.; Pounder R.E.; Samloff I.M.
University Department of Medicine, Royal Free Hospital, School of Medicine, Rowland Hill Street,London NW3 2PF United Kingdom
Alimentary Pharmacology and Therapeutics (ALIMENT. PHARMACOL. THER.) (United Kingdom) 1992, 6/3 (389-394)
CODEN: APTHE ISSN: 0269-2813
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

DRUG DESCRIPTORS:
...*therapy--dt; *bismuth derivative--drug combination--cb; *metronidazole --drug therapy--dt; *metronidazole--drug combination--cb; *pepsinogen i --endogenous compound--ec; *pepsinogen ii--endogenous compound--ec
MEDICAL DESCRIPTORS:
*bacterial infection--drug therapy--dt; *helicobacter pylori
...CAS REGISTRY NO.: 443-48-1 (metronidazole); 92228-49-4 (pepsinogen i); 61536-72-9...

5/K/64 (Item 64 from file: 73)
DIALOG(R)File 73:EMBASE

04943397 EMBASE No: 1992083613
Relationship of Helicobacter pylori to serum pepsinogens in an asymptomatic Japanese population
Asaka M.; Kimura T.; Kudo M.; Takeda H.; Mitani S.; Miyazaki T.; Miki K.; Graham D.Y.
Veterans Affairs Medical Ctr., 2002 Holcombe Boulevard,Houston, TX 77030 United States
Gastroenterology (GASTROENTEROLOGY) (United States) 1992, 102/3 (760-766)
CODEN: GASTA ISSN: 0016-5085
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

DRUG DESCRIPTORS:
*biological marker--endogenous compound--ec; *immunoglobulin g antibody --endogenous compound--ec; *pepsinogen i --endogenous compound--ec; * pepsinogen ii--endogenous compound--ec
MEDICAL DESCRIPTORS:

*atrophic gastritis--etiology--et; ***helicobacter pylori*** ; *gastritis --etiology--et
CAS REGISTRY NO.: 92228-49-4 (**pepsinogen i**); 61536-72-9...

5/K/65 (Item 65 from file: 73)
DIALOG(R)File 73:EMBASE

04930852 EMBASE No: 1992071068
Effect of helicobacter pylori on serum pepsinogen I and plasma gastrin in duodenal ulcer patients
Chittajallu R.S.; Dorrian C.A.; Ardill J.E.S.; McColl K.E.L.
Department of Medicine, Western Infirmary, Glasgow G11 6NT United Kingdom
Scandinavian Journal of Gastroenterology (SCAND. J. GASTROENTEROL.) (Norway) 1992, 27/1 (20-24)
CODEN: SJGRA ISSN: 0036-5521
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

DRUG DESCRIPTORS:
*gastrin--endogenous compound--ec; ***pepsinogen i*** --endogenous compound--ec
MEDICAL DESCRIPTORS:
helicobacter pylori ; *gastrin blood level
CAS REGISTRY NO.: 9002-76-0 (gastrin); 92228-49-4 (**pepsinogen i**)

5/K/66 (Item 66 from file: 73)
DIALOG(R)File 73:EMBASE

04836392 EMBASE No: 1991331128
Helicobacter pylori in dyspeptic patients: Quantitative association with severity of gastritis, intragastric pH, and serum gastrin concentration
Karttunen T.; Niemela S.; Lehtola J.
Dept. of Pathology, University of Oulu, Kajaanintie 52 D,SF-90220 Oulu Finland
Scandinavian Journal of Gastroenterology, Supplement (SCAND. J. GASTROENTEROL. SUPPL.) (Norway) 1991, 26/186 (124-134)
CODEN: SJGSB ISSN: 0085-5928
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

DRUG DESCRIPTORS:
gastrin--drug concentration--cr; gastrin--endogenous compound--ec; **pepsinogen i** --drug concentration--cr; **pepsinogen i** --endogenous compound --ec
MEDICAL DESCRIPTORS:
helicobacter pylori ; *disease severity; *dyspepsia; *gastrin blood level ; *gastritis; *stomach ph
CAS REGISTRY NO.: 9002-76-0 (gastrin); 92228-49-4 (**pepsinogen i**)

5/K/69 (Item 69 from file: 73)
DIALOG(R)File 73:EMBASE

04623565 EMBASE No: 1991117608
Serum pepsinogen I in duodenal ulcer
Kolster J.
Hospital Gonzalez Plaza, Universidad de Carabobo, Carabobo Venezuela
GEN (GEN) (Venezuela) 1990, 44/2 (191-198)
CODEN: GENCA ISSN: 0016-3503
DOCUMENT TYPE: Journal; Review
LANGUAGE: SPANISH SUMMARY LANGUAGE: ENGLISH

DRUG DESCRIPTORS:
...*pd; *histamine h2 receptor antagonist--drug therapy--dt; *omeprazole --pharmacology--pd; *omeprazole--drug therapy--dt; ***pepsinogen i** --pharmacology--pd; ***pepsinogen i** --endogenous compound--ec; *ranitidine --pharmacology--pd; *ranitidine--drug therapy--dt
MEDICAL DESCRIPTORS:
acid secretion; **helicobacter pylori** ; genetic marker; human; review
...CAS REGISTRY NO.: 95510-70-6 (omeprazole); 92228-49-4 (**pepsinogen i**); 66357-35-5...

5/K/70 (Item 70 from file: 73)
DIALOG(R)File 73:EMBASE

04018929 EMBASE No: 1989187971
Serum pepsinogen I and IgG antibody to Campylobacter pylori in non-specific abdominal pain in childhood
Oderda G.; Vaira D.; Holton J.; Dowsett J.F.; Ansaldi N.
Pediatric Gastroenterology Section, University of Torino, Torino Italy
Gut (GUT) (United Kingdom) 1989, 30/7 (912-916)
CODEN: GUTTA ISSN: 0017-5749
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

DRUG DESCRIPTORS:
*immunoglobulin g antibody; ***pepsinogen i** ; *urease
MEDICAL DESCRIPTORS:
*abdominal pain--etiology--et; ***helicobacter pylori** ; *immunoglobulin blood level
CAS REGISTRY NO.: 92228-49-4 (**pepsinogen i**); 9002-13-5 (urease)

5/K/71 (Item 71 from file: 73)
DIALOG(R)File 73:EMBASE

03920265 EMBASE No: 1989089258
Amoxycillin plus tinidazole for campylobacter pylori gastritis in children: Assessment by serum IgG antibody, pepsinogen I, and gastrin levels
Oderda G.; Holton J.; Altare F.; Vaira D.; Ainley C.; Ansaldi N.
Paediatric Gastroenterology Section, University of Turin, Turin Italy
Lancet (LANCET) (United Kingdom) 1989, 1/8640 (690-692)
CODEN: LANCA ISSN: 0140-6736
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

DRUG DESCRIPTORS:
*bacterium antibody; *gastrin; ***pepsinogen i** ; *amoxicillin--drug therapy --dt; *amoxicillin--drug combination--cb; *tinidazole--drug therapy--dt; * tinidazole--drug...
MEDICAL DESCRIPTORS:
***helicobacter pylori** ; *gastritis--diagnosis--di
CAS REGISTRY NO.: 9002-76-0 (gastrin); 92228-49-4 (**pepsinogen i**);

5/9/2 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

10840015 EMBASE No: 2000321262
Evaluation of blood tests to predict normal gastric mucosa
Oksanen A.; Sipponen P.; Miettinen A.; Sarna S.; Rautelin H.
Dr. H. Rautelin, Dept. of Bacteriology and Immunology, University of Helsinki, P.O. Box 21, FIN-00014 Helsinki Finland
Scandinavian Journal of Gastroenterology (SCAND. J. GASTROENTEROL.) (Norway) 2000, 35/8 (791-795)
CODEN: SJGRA ISSN: 0036-5521
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 18

Background: To determine the accuracy of blood tests in predicting normal gastric mucosa confirmed by histological examination of gastric biopsy specimens. Methods: In total, 207 consecutive patients referred for upper endoscopy were included. Two biopsy specimens each from the antrum and corpus were assessed histologically for the presence of Helicobacter pylori, gastritis, and atrophy. Serum samples were studied for H. pylori antibodies by enzyme immunoassay (Pyloriset EIA-G and EIA-A) and by a rapid latex agglutination test (Pyloriset Dry); pepsinogen I was measured by an immunoenzymometric assay (Gastroset PGI), gastrin by radioimmunoassay, and parietal cell antibodies by indirect immunofluorescence. Results: In 101 (49%) of 207 patients, the gastric mucosa on histologic examination was normal. In the 63 patients aged 45 years or less, H. pylori IgG serology was negative in all 47 patients with normal gastric mucosa and none had low serum pepsinogen I levels. Among 144 patients over age 45 years, 72 had negative H. pylori IgG serology. Combining the serum pepsinogen I assay with the results of H. pylori IgG serology, 12 patients with normal serology but low serum pepsinogen I were found. Thus, 60 patients, 52 of whom showed normal gastric histology, had normal IgG serology and serum pepsinogen I. In the remaining eight patients with normal blood tests, the histologic changes were very mild. Conclusions: Although negative H. pylori IgG serology alone in younger patients, and in combination with normal serum pepsinogen I levels in older patients, reliably predicted the presence of normal gastric mucosa, gastroscopy is still recommended for patients over 45 years.

DEVICE BRAND NAME/MANUFACTURER NAME: Pyloriset EIA-G; Pyloriset EIA-A; Pyloriset Dry; Gastroset PGI
DRUG DESCRIPTORS:
immunoglobulin G antibody; parietal cell antibody; **pepsinogen I**
MEDICAL DESCRIPTORS:
*gastritis--diagnosis--di; *gastritis--etiology--et; *stomach mucosa; * serology
stomach disease--diagnosis--di; stomach disease--etiology--et; gastroscopy; **Helicobacter pylori** ; stomach biopsy; histology; antibody titer; enzyme immunoassay; radioimmunoassay; immunofluorescence; human; nonhuman; major clinical study; article; priority journal
CAS REGISTRY NO.: 92228-49-4 (**pepsinogen I**)
SECTION HEADINGS:
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
048 Gastroenterology

5/9/3 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

10723429 EMBASE No: 2000212521
Long-term effect of Helicobacter pylori infection on serum pepsinogens

Kikuchi S.; Kurosawa M.; Sakiyama T.; Tenjin H.; Miki K.; Wada O.; Inaba Y.
S. Kikuchi, Department of Public Health, Aichi Medical University, 21 Aza Karimata, Nagakute-cho, Aichi 480-1195 Japan
AUTHOR EMAIL: kikuchis@aichi-med-u.ac.jp
Japanese Journal of Cancer Research (JPN. J. CANCER RES.) (United Kingdom) 2000, 91/5 (471-476)
CODEN: JJCRE ISSN: 0910-5050
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 21

Serum pepsinogen values are markers of gastric mucosal status and of gastric cancer risk. The effect of Helicobacter pylori infection and sibship size on change of serum pepsinogen values over a seven-year span was investigated. Data from 2584 subjects with phlebotomy were analyzed both in 1989 and in 1996. The subjects were classified by H. pylori serology and sibship size (1-3 vs. 4 and more). Pepsinogen I (PG I) to II (PG II) ratio in '96 minus that in '89 was defined as DeltaPG I/II and compared among the groups. DeltaPG I/II was lower and decrease of PG I/II was more frequent among H. pylori-positive subjects than among negative subjects. The difference was owing to a decrease of PG I in all subjects and owing to an increase of PG II in those not younger than 30 years in '89. In H. pylori-positive subjects, those with a larger sibship size showed lower DeltaPG I/II and higher frequency of PG I/II decline. H. pylori infection exerts a reducing effect on PG I/II during the seven-year span. The effect of H. pylori is stronger among those with a larger sibship size, who are expected to have been infected with H. pylori in childhood. Inducing atrophy of gastric mucosa, which is reflected by a decline of PG I/II, may be one of the mechanisms through which H. pylori elevates the risk of gastric cancer.

DRUG DESCRIPTORS:
*pepsinogen--endogenous compound--ec
pepsinogen I --endogenous compound--ec; pepsinogen II--endogenous compound --ec
MEDICAL DESCRIPTORS:
Helicobacter pylori ; *Gram negative infection
stomach mucosa; stomach cancer; cancer risk; phlebotomy; serology; follow up; sibling; human; male; female; major clinical study; controlled study; adult; article; priority journal
CAS REGISTRY NO.: 9001-10-9 (pepsinogen); 92228-49-4 (**pepsinogen I**); 61536-72-9, 95829-35-9 (pepsinogen II
SECTION HEADINGS:
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
016 Cancer
048 Gastroenterology

1998:372881 CAPLUS
DN 129:39461
TI Diagnosis of **Helicobacter pylori** infection. Serum antibody, **pepsinogen**, and urea breath test
AU Goto, Akira; Fujimori, Kazuya; Kaneko, Taimei; Akamatsu, Taiji
CS 2nd Dep. Intern. Med., Shinshu Univ., Matsumoto, 390, Japan
SO Nippon Naika Gakkai Zasshi (1998), 87(5), 863-867
CODEN: NNGAAS; ISSN: 0021-5384
PB Nippon Naika Gakkai
DT Journal; General Review
LA Japanese

5/K/24 (Item 24 from file: 73)
DIALOG(R)File 73:EMBASE

07076845 EMBASE No: 1997358708
Antigastric autoantibodies in Helicobacter pylori infection: Implications of histological and clinical parameters of gastritis
Faller G.; Steininger H.; Kranzlein J.; Maul H.; Kerkau T.; Hensen J.; Hahn E.G.; Kirchner T.
Dr. G. Faller, Institute of Pathology, University of Erlangen-Nurnberg, Krankenhausstrasse 8-10, D-91054 Erlangen Germany
Gut (GUT) (United Kingdom) 1997, 41/5 (619-623)
CODEN: GUTTA ISSN: 0017-5749
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 38

DRUG DESCRIPTORS:
*epitope--endogenous compound--ec; *gastrin--endogenous compound--ec; *parietal cell antibody--endogenous compound--ec; ***pepsinogen i** --endogenous compound--ec; *pepsinogen ii--endogenous compound--ec
MEDICAL DESCRIPTORS:
*gastritis; *gram negative infection; ***helicobacter pylori**
CAS REGISTRY NO.: 9002-76-0 (gastrin); 92228-49-4 (**pepsinogen i**); 61536-72-9...

5/K/26 (Item 26 from file: 73)
DIALOG(R)File 73:EMBASE

06997555 EMBASE No: 1997283757
Changes in gastrin and serum pepsinogens in monitoring of Helicobacter pylori response to therapy
Perez-Paramo M.; Albillos A.; Calleja J.L.; Salas C.; Marin M.D.C.; Marcos M.L.; Cacho G.; Escartin P.; Ortiz-Berrocal J.
Dr. A. Albillos, Department of Gastroenterology, Clinica Puerta de Hierro, San Martin de Porres, 4, 28035 Madrid Spain
Digestive Diseases and Sciences (DIG. DIS. SCI.) (United States) 1997, 42/8 (1734-1740)
CODEN: DDSCD ISSN: 0163-2116
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 22

DRUG DESCRIPTORS:
...*endogenous compound--ec; *omeprazole--drug therapy--dt; *omeprazole --drug dose--do; *omeprazole--drug combination--cb; ***pepsinogen i** --endogenous compound--ec; *pepsinogen ii--endogenous compound--ec
MEDICAL DESCRIPTORS:
...*ulcer--drug therapy--dt; *gram negative infection--etiology--et; *gram negative infection--drug therapy--dt; ***helicobacter pylori**
...CAS REGISTRY NO.: 95510-70-6 (omeprazole); 92228-49-4 (**pepsinogen i**); 61536-72-9...

5/K/30 (Item 30 from file: 73)
DIALOG(R)File 73:EMBASE

06756211 EMBASE No: 1997037697
Serum pepsinogen I levels and acid secretion in Helicobacter pylori associated enlarged fold gastritis
Yasunaga Y.; Shinomura Y.; Kanayama S.; Miyazaki Y.; Palacios J.J.B.; Matsuzawa Y.
Dr. Y. Yasunaga, Second Department Internal Medicine, Osaka University

Medical School, 2-2 Yamadaoka, Suita, Osaka 565 Japan
Italian Journal of Gastroenterology (ITAL. J. GASTROENTEROL.) (Italy) 1996, 28/8 (457-461)
CODEN: ITJGD ISSN: 0392-0623
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 35

DRUG DESCRIPTORS:
***pepsinogen i** --endogenous compound--ec
MEDICAL DESCRIPTORS:
...level; clinical article; female; gram negative infection--drug therapy --dt; gram negative infection--etiology--et; **helicobacter pylori** ; human; male; stomach parietal cell
CAS REGISTRY NO.: 92228-49-4 (**pepsinogen i**); 10361-44-1...

5/K/31 (Item 31 from file: 73)
DIALOG(R)File 73:EMBASE

06738461 EMBASE No: 1997019932
Percentage changes in serum pepsinogens are useful as indices of eradication of Helicobacter pylori
Furuta T.; Kaneko E.; Baba S.; Arai H.; Futami H.
Dr. T. Furuta, First Department of Medicine, Hamamatsu Univ. School of Medicine, 3600 Handa-cho, Hamamatsu 431-31 Japan
American Journal of Gastroenterology (AM. J. GASTROENTEROL.) (United States) 1997, 92/1 (84-88)
CODEN: AJGAA ISSN: 0002-9270
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 20

DRUG DESCRIPTORS:
***pepsinogen i** --endogenous compound--ec; *pepsinogen ii--endogenous compound--ec
MEDICAL DESCRIPTORS:
*gram negative infection--diagnosis--di; *gram negative infection--drug therapy--dt; ***helicobacter pylori** ; *serum
CAS REGISTRY NO.: 92228-49-4 (**pepsinogen i**); 61536-72-9...

5/K/39 (Item 39 from file: 73)
DIALOG(R)File 73:EMBASE

06388260 EMBASE No: 1996047954
Verification of decreased basal and stimulated serum pepsinogen-I levels is a useful non-invasive method for determining the success of eradication therapy for Helicobacter pylori
Gisbert J.P.; Boixeda D.; Vila T.; De Rafael L.; Redondo C.; Canton R.; Martin de Argila C.
C/Lagasca 13,E-28001 Madrid Spain
Scandinavian Journal of Gastroenterology (SCAND. J. GASTROENTEROL.) (Norway) 1996, 31/2 (103-110)
CODEN: SJGRA ISSN: 0036-5521
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

DRUG DESCRIPTORS:
*metronidazole--drug therapy--dt; *metronidazole--drug combination--cb; ***pepsinogen i** --endogenous compound--ec; *tetracycline--drug therapy--dt; *tetracycline--drug combination--cb
MEDICAL DESCRIPTORS:
adult; aged; article; clinical article; clinical trial; controlled study;

drug efficacy; female; **helicobacter pylori** ; human; human tissue; male; priority journal; stomach biopsy
...CAS REGISTRY NO.: 443-48-1 (metronidazole); 92228-49-4 (**pepsinogen i**); 23843-90-5...

5/K/40 (Item 40 from file: 73)
DIALOG(R)File 73:EMBASE

06376091 EMBASE No: 1996040882
Serum pepsinogen I in childhood Helicobacter pylori gastritis: Its relation to mucosal peptic activity
Yahav J.; Oderda G.; Diver-Haber A.; Fradkin A.; Keller N.; Altare F.; Ansaldi N.; Jonas A.
Pediatric Gastroenterology Unit, Chaim Sheba Medical Center,52621 Tel-Hashomer Israel
Israel Journal of Medical Sciences (ISR. J. MED. SCI.) (Israel) 1996, 32/1 (56-59)
CODEN: IJMDA ISSN: 0021-2180
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

DRUG DESCRIPTORS:
*pepsinogen i --endogenous compound--ec
MEDICAL DESCRIPTORS:
...adult; article; child; childhood disease--etiology--et; controlled study ; enzyme activity; enzyme blood level; female; **helicobacter pylori** ; human ; human tissue; major clinical study; male; pepsin secretion; stomach antrum
CAS REGISTRY NO.: 92228-49-4 (**pepsinogen i**); 9001-75-6 (pepsin a)

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File 155:MEDLINE(R) 1966-2000/Dec W4
         (c) format only 2000 Dialog Corporation
File  73:EMBASE 1974-2001/Feb W1
         (c) 2001 Elsevier Science B.V.
?ds

Set     Items   Description
S1      23081   HELICOBACTER PYLORI
S2        190   PEPSINOGEN I
S3         71   S1 AND S2
S4          0   H,K-ATPASE
S5         71   RD S3 (unique items)
?t s5/free/1-10
```

5/6/2 (Item 2 from file: 73)
10840015 EMBASE No: 2000321262
Evaluation of blood tests to predict normal gastric mucosa
2000

5/6/3 (Item 3 from file: 73)
10723429 EMBASE No: 2000212521
Long-term effect of Helicobacter pylori infection on serum pepsinogens
2000

24 26 , 30 , 31 , 39 , 40 , 46 , 49 , 60 , 64

65, 66 , 69 , 70 , 71

DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

10546918 20335634
Antibody enzymes in Helicobacter pylori-associated infection]
Antitela-fermenty pri Helicobacter pylori -assotsiirovannoi infektsii.
Konorev MR
State Medical Institute, Vitebsk, Belarus.
Zhurnal mikrobiologii, epidemiologii, i immunobiologii (RUSSIA) Jan-Feb 2000, (1) p75-9, ISSN 0372-9311 Journal Code: Y9O
Languages: RUSSIAN Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE ; English Abstract

12/3/2
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

10543336 20341254
Noninvasive tests as a substitute for histology in the diagnosis of Helicobacter pylori infection.
Hahn M; Fennerty MB; Corless CL; Magaret N; Lieberman DA; Faigel DO
Division of Gastroenterology, Portland VA Medical Center and Oregon Health Sciences University, OR 97201, USA.
Gastrointestinal endoscopy (UNITED STATES) Jul 2000, 52 (1) p20-6, ISSN 0016-5107 Journal Code: FH8
Languages: ENGLISH
Document type: CLINICAL TRIAL; JOURNAL ARTICLE

12/3/3
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

10455773 20307965
Factors that affect results of the 13C urea breath test in Japanese patients.
Chen X; Haruma K; Kamada T; Sumii M; Komoto K; Yoshihara M; Sumii K; Kajiyama G
Gastrointestinal Unit, First Department of Internal Medicine, University School of Medicine, Hiroshima, Japan.
Helicobacter (UNITED STATES) Jun 2000, 5 (2) p98-103, ISSN 1083-4389 Journal Code: CY4
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/4
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

10422738 20282082
The assessment of the degree of the colonization of the gastric mucosa by Helicobacter pylori and of the gastritis and duodenitis activity in duodenal peptic ulcer in different age groups]
Otsinka stupenia obsimenia slyzovoi obolonky shlunka Helicobacter pylori ta aktyvnost gastrytu i duodenitu pry vyrazkovii khvorobi dvanadtsiatipaloi kyshky v riznykh vikovykh hrupakh.
Soloviova HA
Likars'ka sprava (UKRAINE) Jul 1999, (5) p41-3, ISSN 1019-5297 Journal Code: CIU
Languages: UKRAINIAN Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE ; English Abstract

12/3/5
DIALOG(R)File 155:MEDLINE(R)

(c) format only 2000 Dialog Corporation. All rts. reserv.

10339884 20183029
Diagnosis of Helicobacter pylori infection in patients with atrophic gastritis : comparison of histology, 13C-urea breath test, and serology.
Kokkola A; Rautelin H; Puolakkainen P; Sipponen P; Farkkila M; Haapiainen R; Kosunen TU
Second Dept. of Surgery, Helsinki University Central Hospital, Finland.
Scandinavian journal of gastroenterology (NORWAY) Feb 2000, 35 (2) p138-41, ISSN 0036-5521 Journal Code: UCS
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/6
DIALOG(R)File 155 MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

10271365 20102100
Evaluation of rapid antibody tests for the diagnosis of Helicobacter pylori infection.
Faigel DO; Magaret N; Cordes C; Lieberman DA; Fennerty MB
Department of Medicine, Portland VA Medical Center, Oregon 97201, USA.
American journal of gastroenterology (UNITED STATES) Jan 2000, 95 (1) p72-7, ISSN 0002-9270 Journal Code: 3HE
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/7
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

10229151 20072320
Fingerstick Helicobacter pylori antibody test: better than laboratory serological testing?
Laine L; Knigge K; Faigel D; Magaret N; Marquis SP; Vartan G; Fennerty MB
Department of Medicine, USC School of Medicine, Los Angeles, California 90033, USA.
American journal of gastroenterology (UNITED STATES) Dec 1999, 94 (12) p3464-7, ISSN 0002-9270 Journal Code: 3HE
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/8
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

10229150 20072319
How useful is the detection kit for antibody to Helicobacter pylori in urine (URINELISA) in clinical practice?
Miwa H; Hirose M; Kikuchi S; Murai T; Iwazaki R; Kobayashi O; Takei Y; Ogihara T; Sato N
Department of Gastroenterology, Juntendo University, School of Medicine, Tokyo, Japan.
American journal of gastroenterology (UNITED STATES) Dec 1999, 94 (12) p3460-3, ISSN 0002-9270 Journal Code: 3HE
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/9
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

10083653 97372316
The evaluation of tissue kallikrein in Helicobacter pylori**-associated gastric ulcer disease.**
Naidoo S; Ramsaroop R; Bhoola R; Bhoola KD
Department of Experimental and Clinical Pharmacology, University of Natal Medical School, Durban, South Africa. naidoot@und.med.ac.za
Immunopharmacology (NETHERLANDS) Jun 1997, 36 (2-3) p263-9, ISSN 0162-3109 Journal Code: GY3
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/10
DIALOG(R)File 155:MEDLINE(R)
(c) format only 200 [illegible] reserv.

10070108 99394787
Helicobacter pylori **infection pattern of gastritis, and symptoms in erosive and nonerosive gastroesophageal reflux disease.**
Manes G; Mosca S; Laccetti M; Lombiello M; Balzano A
Dept. of Gastroenterology, Cardarelli Hospital, Naples, Italy.
Scandinavian journal of gastroenterology (NORWAY) Jul 1999, 34 (7) p658-62, ISSN 0036-5521 Journal Code: UCS
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/11
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

10041738 99409131
Behavior of Helicobacter [illegible] **in 3d and 4th age-bracket patients]**
Comportamento di **Helicobacter** pylori in pazienti della III e IV eta.
Daturi R; Matti C; Nicolato A; Giacobone E; Cipolli PL; Romero E; Pannella A; Tattarletti L; Monacasa P; Brunati S; Zambianchi M
Servizio Analisi Microbiologiche, IRCCS Policlinico San Matteo, Pavia.
Minerva chirurgica (ITALY) Jun 1999, 54 (6) p411-4, ISSN 0026-4733 Journal Code: N3I
Languages: ITALIAN Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE English Abstract

12/3/12
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

10001850 99372707
Usefulness of serological [illegible] determinations for confirming eradication of Helicobacter pylori **infection.**
Marchildon P; Bourdon DH; [illegible] Charles C; Doobay R; Passaretti N; Peacock J; Marshall BJ; Peura DA
Enteric Products, Inc., Stony Brook, New York 11790, USA.
American journal of gastroenterology (UNITED STATES) Aug 1999, 94 (8) p2105-8, ISSN 0002-9270 Journal Code: 3HE
Languages: ENGLISH
Document type: CLINICAL TRIAL JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

12/3/13
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

09994980 99306116
Realities of diagnosing Helicobacter pylori **infection in clinical practice: a case for non-invasive indirect methodologies.**
Metz DC; Furth EE; Faigel [illegible] Kroser JA; Alavi A; Barrett DM; Montone K

Department of Pathology and Laboratory Medicine, University of Pennsylvania Medical Center, Philadelphia 19104, USA. Metzda@mail.med.upenn.edu
Yale journal of biology and medicine (UNITED STATES) Mar-Apr 1998, 71 (2) p81-90, ISSN 0044-0086 Journal Code: XR7
Languages: ENGLISH
Document type: CLINICAL TRIAL; JOURNAL ARTICLE

12/3/14
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

09976043 99311132
Prevalence of Cag, VacA antibodies in symptomatic and asymptomatic children with Helicobacter pylori **infection.**
Elitsur Y; Neace C; Wertheimer J; Lawt WE
Department of Pediatrics, Marshall University School of Medicine, Huntington, West Virginia 25701-3655, USA.
Helicobacter (UNITED STATES) Jun 1999, 4 (2) p100-5, ISSN 1083-4389 Journal Code: CY4
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/15
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

09963839 99292015
A close relationship between Helicobacter pylori **infection and gastric xanthoma.**
Isomoto H; Mizuta Y; Inoue K; Murata I; Kiyakawa T; Miyazaki M; Onita K; Takeshima F; Murase K; Shimokawa I; Kohno S
Second Dept. of Internal Medicine, Nagasaki University School of Medicine, Japan.
Scandinavian journal of gastroenterology (NORWAY) Apr 1999, 34 (4) p346-52, ISSN 0036-5521 Journal Code: UCS
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/16
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

09894654 99219298
Evaluation of a new immunochromatographic test for Helicobacter pylori **IgG antibodies in elderly symptomatic patients [see comments]**
Shirin H; Bruck R; Shapiro G; Epshtein Y; Reif S; Zaidel L; Geva D; Avni Y; Halpern Z
Department of Gastroenterology, The E. Wolfson Medical Center, Holon, Israel.
Journal of gastroenterology (JAPAN) Feb 1999, 34 (1) p7-10, ISSN 0944-1174 Journal Code: BWP
Comment in J Gastroenterol 1999 Feb;34(1):145-6
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/17
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

09853779 99151749
Differential Helicobacter pylori **infection rates in two contrasting gastric cancer risk regions in South China. China Gastric Cancer Study Group.**

Wong BC; Lam SK; Ching CK; Hu WH; Kwok E; Ho J; Yuen ST; Gao Z; Chen JS; Lai KC; Ong LY; Chen BW; Wang WH, Jiang XW; Hou XH; Lu JY
University Department of Medicine, The University of Hong Kong, China.
Journal of gastroenterology and hepatology (AUSTRALIA) Feb 1999, 14 (2) p120-5, ISSN 0815-9319 Journal Code: A6J
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/18
DIALOG(R)File 155:MEDLINE(R)

09827307 99165990
CLO antibody assay--can it be an accurate substitute for endoscopy and biopsy?
Mehdi I; Qureshi H; Ahmed F; Molvi G; Alam SE
Pakistan Medical Research Council, Jinnah Postgraduate Medical Centre, Karachi.
JPMA. The Journal of the Pakistan Medical Association (PAKISTAN) Jul 1998, 48 (7) p203-5, ISSN 0030-9982 Journal Code: KGI
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/19
DIALOG(R)File 155:MEDLINE(R)

09788103 99131439
A prospective, multidisciplinary evaluation of premenopausal women with iron-deficiency anemia [see comments]
Kepczyk T; Cremins JE; Long [illegible]ki MJ; Smith LR; McNally PR
Department of Medicine, [illegible] Medical Center, Aurora, Colorado, USA.
American journal of gastroenterology (UNITED STATES) Jan 1999, 94 (1) p109-15, ISSN 0002-9270 Journal Code: 3HE
Comment in Am J Gastroenterol 1999 Jun;94(6):1715
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/20
DIALOG(R)File 155:MEDLINE(R)

09775492 99052819
Autoantibodies reacting with gastric antigens in Helicobacter pylori associated body gastritis in dyspeptic children.
Ierardi E; Francavilla R; [illegible]; [illegible]; Francavilla A
Chair of Gastroenterology, [illegible] of Bari, Italy.
Italian journal of gastroenterology and hepatology (ITALY) Oct 1998, 30 (5) p478-80, Journal Code:
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/21
DIALOG(R)File 155:MEDLINE(R)

09765988 99053746
Serologic detection of Helicobacter pylori infection with cagA-positive strains in [illegible], gastric cancer, and asymptomatic gastritis.
Miehlke S; Go MF; [illegible]m JL; [illegible]DY; Kuipura N.
The Department of Medicine, Veterans Affairs Medical Center (111D), Baylor College of Medicine, Houston, TX 77030, USA.
Journal of gastroenterology (JAPAN) 1998, 33 Suppl 10 p18-21, ISSN

0944-1174 Journal Code: BWP
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/22
DIALOG(R)File 155:MEDLINE(R)
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09678643 98344635
Etiologic aspects of chronic urticaria.
Liutu M; Kalimo K; Uksila J; Kalimo H
Department of Dermatology, Medical Microbiology, and Pathology, University of Turku, Finland.
International journal of dermatology (UNITED STATES) Jul 1998, 37 (7) p515-9, ISSN 0011-9059 Journal Code: GRP
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/23
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

09662554 98436530
Blood tests in the management of Helicobacter pylori **infection. Italian** Helicobacter pylori Study Group.
Vaira D; Holton J; Menegatti M; Landi F; Ricci C; Ali A; Gatta L; Farinelli S; Acciardi C; Massa V; Miglioli M
Department of Internal Medicine, University of Bologna, Italy.
Gut (ENGLAND) Jul 1998, 43 Suppl 1 pS38-46, ISSN 0017-5749 Journal Code: FVT
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

12/3/24
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

09563472 98349343
Role of seroconversion in confirming cure of Helicobacter pylori **infection.**
Feldman M; Cryer B; Lee E; Peterson WL
Department of Internal Medicine, University of Texas Health Science Center at Dallas, USA. [illegible]@va.gov
JAMA (UNITED STATES) 22-29 Jul 1998, 280 (4) p363-5, ISSN 0098-7484 Journal Code: KFR
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/25
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

09555787 98281740
Positive result by serology indicates active Helicobacter pylori **infection in patients with atrophic gastritis.**
Kokkola A; Rautelin H; Puolakkainen P; Sipponen P; Farkkila M; Haapiainen R; Kosunen TU
Second Department of Surgery, Helsinki University Central Hospital, Finland.
Journal of clinical microbiology (UNITED STATES) Jun 1998, 36 (6) p1808-10, ISSN 0095-1137 Journal Code: HSH
Languages: ENGLISH
Document type: CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

12/3/26
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

09484591 98201914
Evaluation of Pyloriset Screen, a rapid whole-blood diagnostic test for Helicobacter pylori infection.
Oksanen A; Veijola L; Sipponen P; Schauman KO; Rautelin H
Herttoniemi Municipal Hospital, Helsinki, Finland.
Journal of clinical microbiology (UNITED STATES) Apr 1998, 36 (4) p955-7, ISSN 0095-1137 Journal Code: HSH
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/27
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

09419389 98140657
Comparison of rapid office-based serology with formal laboratory-based ELISA testing for diagnosis of Helicobacter pylori gastritis.
Kroser JA; Faigel DO; Furth EE; Metz DC
Department of Pathology and Laboratory Medicine, University of Pennsylvania Medical Center, Philadelphia 19104, USA.
Digestive diseases and sciences (UNITED STATES) Jan 1998, 43 (1) p103-8, ISSN 0163-2116 Journal Code: EAD
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/28
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

09402375 98127765
Anti- Helicobacter pylori **specific** antibody **immunohistochemistry improves the diagnostic accuracy of** Helicobacter pylori **in biopsy specimen from patients treated with triple therapy.**
Marzio L; Angelucci D; Grossi L; Isodoro MG; Di Campli E; Cellini L
Institute of Fisiopatologia Medica, Universita G. D'Annunzio, Chieti, Italy.
American journal of gastroenterology (UNITED STATES) Feb 1998, 93 (2) p223-6, ISSN 0002-9270 Journal Code: 3HI
Languages: ENGLISH
Document type: JOURNAL

12/3/29
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

09377359 98060415
Use of serum-specific immunoglobulins A and G for detection of Helicobacter pylori infection in patients with chronic gastritis by immunoblot analysis.
Karvar S; Karch H; Frosch M; Burghardt W; Gross U
Clinic for Internal Medicine, University of Wurzburg, Germany.
Journal of clinical microbiology (UNITED STATES) Dec 1997, 35 (12) p3058-61, ISSN 0095-1137 Journal Code: HSH
Languages: ENGLISH
Document type: JOURNAL ARTICLE

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How should Helicobacter **pylori infection be diagnosed?**
Megraud F
Laboratoire de Bacteriologie, Hopital Pellegrin, Universite de Bordeaux, France.
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Languages: ENGLISH
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09310734 97465294
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Abdullah AM; Gad el Rab M; el Ayed I; al Mazyad AS; al Sanie A
Faculty of Medicine, King Saud University, Riyadh, Saudi Arabia.
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09261930 97452309
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Institut fur Medizinische Mikrobiologie und Hygiene, Universitat zu Koln, Germany.
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Chen JJ; Yang ZX; Jiang XG
Institute of Epidemiology & Microbiology, Chinese Academy of Preventive Medicine, Beijing.
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Hayashi S; Sugiyama T; Hisano K; ...wakawa T; Kurokawa I; Yachi A; Isogai H; Isogai E; Yokota K; Hirai Y; ...um K; Fujii N
Department of Microbiology, Sapporo Medical University, Japan.
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Sinusoidal'nye modulirovannye toki v terapii khronicheskogo gastroduodenita u detei.
Bukanovich OV; Khan MA; Chistova ; Sheliapina VV; Shavrov AA; Klochkov SA
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Variability in serum pepsinogen levels in an asymptomatic population.
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Department of Surgery, School of Postgraduate Medicine, Keele University, Staffordshire, UK.
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Department of Infectious Diseases, Hvidovre University Hospital, Copenhagen, Denmark.
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Languages: ENGLISH
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Department of Pediatrics, Seoul National University College of Medicine, Korea.
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Clinica Industrial, Caracas, Corpoven.
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Languages: SPANISH Summary Languages: ENGLISH
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Department of Gastroenterology and Hepatology, University Hospital Leiden, The Netherlands.
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Department of Pediatric Gastroenterology and Nutrition, James Whitcomb Riley Hospital for Children, Indiana University School of Medicine, Indianapolis 46202-5225, USA.
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Department of Clinical Microbiology, National University, Rigshospitalet, Copenhagen, Denmark.
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Languages: ENGLISH
Document type: JOURNAL ARTICLE

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2nd Department of Internal Medicine, Nippon Medical School, Tokyo, Japan.
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Languages: JAPANESE Summary Languages: ENGLISH
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Department of Medicine, University of Southern California School of Medicine, Los Angeles.
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08153793 95326721
Comparative evaluation of culture techniques and ELISA test in detection of Helicobacter pylori **infection**
Porownawcza ocena posiewu i testu ELISA w wykrywaniu zaka.ANG.zenia **Helicobacter pylori**
Sito E; Karczewska E; Krzeha[illegible]; Oleksy J; Nowak-Sadzikowska J; Maj A
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Neue Wiener Privatklinik, Wien.
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Takagi A; Ohta U; [illegible]hi T; [illegible]K; Kobayashi H; Harasawa S; Miwa T; Kamiya S
Department of [illegible]tol Medi[illegible] Tokai University School of Medicine.
Nippon Shokakibyo Gakkai [illegible](JAPAN) Aug 1994, 91 (8) p1277-82, ISSN 0446-6586 Journal
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DIALOG(R)File 155:MEDLINE(R)
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The humoral im[illegible]e to Helicobacter pylori infection in children with recurrent abdominal
Andersen LP; Wewer AV; Ch[illegible]FM; Tvede M; Hansen JP; Henriksen FW; Krasilnikoff PA
Department of Clinical Mic[illegible]ogy, National University, Rigshospitalet, Copenhagen, Denmark.
APMIS (DENMARK) Jan 1994 [illegible]-64, ISSN 0903-4641 Journal Code: AMS
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Best LM; Veldhuyzen van Zanten SJ; Sherman PM; Bezanson GS
Department of Microbiology, Victoria General Hospital, Halifax, Nova Scotia, Canada.
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Postgraduate medical journal (ENGLAND) Jun 1993, 69 (812) p456-60, ISSN 0032-5473 Journal Code: PMX
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Section of Gastroenterology, Veterans Affairs Medical Center, Martinez, California.
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Department of Microbiology, Medical Academy, WrocLaw, Poland.
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Document type: CLINICAL TRIAL; JOURNAL ARTICLE

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07694013 94085142
Role of Helicobacter pylori serology in evaluating treatment success.
Cutler A; Schubert A; Schubert T
Division of Gastroenterology, Henry Ford Hospital, Detroit, Michigan.
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07617211 93382585
Costs and effectiveness of diagnosis and treatment of patients with dyspepsia, determined with the aid of a computer model (see comments)]
Kosten en effectiviteit van diagnostiek en behandeling van patienten met dyspepsie, bepaald met een computermodel.
Schipper CK; Rutten FF; Lofgeld R
Erasmus Universiteit, Instituut voor Medische Technology Assessment, Rotterdam.
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Document type: JOURNAL ARTICLE ; English Abstract

12/3/75
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

07593273 93343747
Gastric syphilis. Primary diagnosis by gastric biopsy: report of four cases.
Fyfe B; Poppiti RJ Jr; Robertson MJ
Arkadi M. Rywlin Department of Pathology and Laboratory Medicine, Mount Sinai Medical Center, Miami, FL 33140.
Archives of pathology & laboratory medicine (UNITED STATES) Aug 1993, 117 (8) p820-3, ISSN 0003-9985 Journal Code: 79Z
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/76
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

07589200 93332059

Apparent reversal of early gastric mucosal atrophy after triple therapy for Helicobacter pylori.
Borody TJ; Andrews P; Jankiewicz E; Ferch N; Carroll M
Centre for Digestive Diseases, NSW, Australia.
American journal of gastroenterology (UNITED STATES) Aug 1993, 88 (8) p1266-8, ISSN 0002-9270 Journal Code: 3HE
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/77
DIALOG(R)File 155:MEDLINE(R)

07569772 93301099
Diagnosis **of** Helicobacter pylori **infection by using pyloriset EIA-G and EIA-A for detection of** serum **immunoglobulin G (IgG) and IgA antibodies [published erratum appears in J Clin Microbiol 1993 Sep;31(9):2556]**
Granberg C; Mansikka A; Lahtonen Kujari H; Gronfors R; Nurmi H; Raiha I; Stahlberg MR; Leino R
Orion Corporation, Orion Diagnostica, Espoo, Finland.
Journal of clinical microbiology (UNITED STATES) Jun 1993, 31 (6) p1450-3, ISSN 0095-1137 Journal Code: HSH
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/78
DIALOG(R)File 155:MEDLINE(R)

07541184 93251516
Antigens for the ELISA test for serodiagnosis of Helicobacter pylori **infection]**
Antigeny pre ELISA test na serodiagnostiku infekcie **Helicobacter pylori**.
Buchvald D; Buchvaldova P
Ustav imunologie Univerzity Komenskeho, Bratislava.
Ceskoslovenska epidemiologie, mikrobiologie, imunologie (CZECHOSLOVAKIA) Mar 1993, 42 (1) p16-21, ISSN 0009-0522 Journal Code: CSH
Languages: SLOVAK Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE ; English Abstract

12/3/79
DIALOG(R)File 155:MEDLINE(R)

07424520 92165002
Relationship of Helicobacter pylori **to serum pepsinogens in an asymptomatic Japanese population.**
Asaka M; Kimura T; Kudo M; Takeda H; Mitani S; Miyazaki T; Miki K; Graham DY
Third Department of Internal Medicine, Hokkaido University School of Medicine, Sapporo, Japan.
Gastroenterology (UNITED STATES) Mar 1992, 102 (3) p760-6, ISSN 0016-5085 Journal Code: FH3
Contract/Grant No.: DK 39919, DK, NIDDK
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/80
DIALOG(R)File 155:MEDLINE(R)

07419107 91318374
Serum immune responses to Helicobacter pylori **in children:**

epidemiologic and clinical applications.
De Giacomo C; Lisato L; Negrini R; Licardi G; Maggiore G
Clinica Pediatrica dell'Universita di Pavia, IRCCS Policlinico S. Matteo, Italy.
Journal of pediatrics (UNITED STATES) Aug 1991, 119 (2) p205-10, ISSN 0022-3476 Journal Code: JLZ
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/81
DIALOG(R)File 155:MEDLINE(R)
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07413463 91064694
Helicobacter pylori and gastric **carcinoma. Serum** antibody **prevalence in populations with** increasing **cancer risks.**
Correa P; Fox J; Fontham E; Ruiz B; Lin YP; Zavala D; Taylor N; Mackinley D; de Lima E; Portilla H; et al
Department of Pathology, Louisiana State University Medical Center, New Orleans 70112.
Cancer (UNITED STATES) Dec 15 1990, 66 (12) p2569-74, ISSN 0008-543X Journal Code: CLZ
Contract/Grant No.: CA-28842, CA, NCI; AI-25590, AI, NIAID; AI25631, AI, NIAID; +
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/82
DIALOG(R)File 155:MEDLINE(R)
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07406097 93195430
Clinical significance of anti-Helicobacter pylori antibody **in the** diagnosis **of** Helicobacter pylori **infection in chronic** gastritis]
Negayama K; Terada S; Kawamoto I
Department of Clinical Laboratory, Kagawa Medical School.
Kansenshogaku zasshi (JAPAN) Nov 1992, 66 (11) p1597-8, ISSN 0387-5911 Journal Code:
Languages: JAPANESE
Document type: JOURNAL ARTICLE

12/3/83
DIALOG(R)File 155:MEDLINE(R)
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07357471 91178057
Evaluation of an enzyme immunoassay **for specific detection of immunoglobulin G** antibodies **against** Helicobacter pylori, **and antigenic cross-reactivity by** Western blot **with** Helicobacter pylori **and Campylobacter jejuni.**
Faulde M; Putzker M; Mertes T;
Department of Medical Microbiology, Ernst-Rodenwaldt-Institute, Koblenz, Federal Republic of Germany.
Journal of clinical microbiology (UNITED STATES) Feb 1991, 29 (2) p323-7, ISSN 0095-1137 Journal Code: HS
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/84
DIALOG(R)File 155:MEDLINE(R)
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07336365 90197249
Helicobacter pylori **in Italian children with chronic diarrhoea and malnutrition.**

Sullivan PB; Thomas JE; Wight DG; Neale G; Eastham EJ; Corrah T; Lloyd-Evans N; Greenwood BM
MRC Laboratories, Fajara, The Gambia.
Archives of disease in childhood (ENGLAND) Feb 1990, 65 (2) p189-91, ISSN 0003-9888 Journal Code: 6XG
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/85
DIALOG(R)File 155:MEDLINE(R)

07169161 93071396
Helicobacter pylori, gastritis and **ulcers in pediatrics.**
Judd RH
University of Wisconsin Hospital, Madison.
Advances in pediatrics (UNITED STATES) 1992, 39 p283-306, ISSN 0065-3101 Journal Code: 200
Languages: ENGLISH
Document type: HISTORICAL ARTICLE; JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

12/3/86
DIALOG(R)File 155:MEDLINE(R)

07145544 93033608
Detection of Helicobacter pylori **infections by** antibody **determination]**
Nachweis von **Helicobacter** -pylori -Infektionen durch Antikorperbestimmung.
Briedigkeit H; Montag T; Spiridonow PS; Sielaff F; Wack R; Held C; Hantke C
Universitatsklinik fur Innere Medizin Theodor Brugsch, Medizinischen Fakultat, Humboldt-Universitat zu Berlin.
Zeitschrift fur arztliche Fortbildung (GERMANY) Sep 10 1992, 86 (17) p869-72, ISSN 0044-2178 Journal Code: XS6
Languages: GERMAN
Document type: JOURNAL ARTICLE

12/3/87
DIALOG(R)File 155:MEDLINE(R)

07084474 92351036
Serodiagnosis of Helicobacter pylori**-associated** gastritis **with a monoclonal** antibody **competitive enzyme-linked immunosorbent assay.**
Negrini R; Zanella I; [illegible] A; Soles C; Verardi R; Ghielmi S; Albertini A; Sangaletti O; Lazzaroni M; Bianchi Porro G
Institute of Chemistry, School of Medicine, University of Brescia, Italy.
Scandinavian journal of gastroenterology (NORWAY) Jul 1992, 27 (7) p599-605, ISSN 0036-5521 Journal Code: UCS
Languages: ENGLISH
Document type: JOURNAL A.

12/3/88
DIALOG(R)File 155:MEDLINE(R)

06878854 92206121
The role of serology in the diagnosis of Helicobacter **(Campylobacter)** pylori **infection]**
Interets de la serologie dans la detection de l'infection a **Helicobacter (Campylobacter) pylori**

Fannes F; Pierard P; Baise E; Hulin G
Laboratoire de Recherche et Developpement, Wavre, Limal.
Acta gastro-enterologica belgica (BELGIUM) Sep-Dec 1991, 54 (5-6) p368-74, ISSN 0001-5644 Journal Code: ONY
Languages: FRENCH Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL ; English Abstract

12/3/89
DIALOG(R)File 155:MEDLINE(R)

06843074 92105332
Characterization of [illegible] **species-specific 19-kilodalton outer membrane protein from** [illegible] **pylori by using a monoclonal** antibody.
Drouet EB; Denoyel GA; [illegible]de M; Wallano E; Andujar M; de Montclos HP
Division of Infectious Diseases, Institut Pasteur de Lyon, France.
Journal of clinical microbiology (UNITED STATES) Aug 1991, 29 (8) p1620-4, ISSN 0095-1137 Journal Code: HSH
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/90
DIALOG(R)File 155:MEDLINE(R)

06835128 92090747
Long term serological surveillance after treatment of Helicobacter pylori **infection.**
Veenendaal RA; Pena AS; Meuwissen JL; Endtz HP; van der Est MM; van Duijn W; Eulderink F; Kreuning J; Lamers CB
Department of Gastroenterology, Leiden University Hospital, The Netherlands.
Gut (ENGLAND) Nov 1991, 32 (11) p1291-4, ISSN 0017-5749 Journal Code: FVT
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/91
DIALOG(R)File 155:MEDLINE(R)

06793852 92022172
Diagnosis **of** gastritis caused by Helicobacter pylori **in children by means of an ELISA.**
Czinn SJ; Carr HS; Speck WT
Department of Pediatrics, School of Medicine, Case Western Reserve University, Cleveland, Ohio.
Reviews of infectious diseases (UNITED STATES) Jul-Aug 1991, 13 Suppl 8 pS700-3, ISSN 0162-0886 Journal Code: SXN
Contract/Grant No.: AI 25819, AI, NIAID
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/92
DIALOG(R)File 155:MEDLINE(R)

06765609 91367230
Helicobacter pylori **infection and gastric carcinoma among Japanese Americans in Hawaii [see comments].**
Nomura A; Stemmermann GN; Chyou PH; Kato I; Perez-Perez GI; Blaser MJ
Japan-Hawaii Cancer Study, Kuakini Medical Center, Honolulu 96817.

New England journal of medicine (UNITED STATES) Oct 17 1991, 325 (16) p1132-6, ISSN 0028-4793 Journal Code: NOW
Contract/Grant No.: R01-CA-33644, CA, NCI
Comment in N Engl J Med 1991 Oct 17;325(16):1170-1
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/93
DIALOG(R)File 155:MEDLINE(R)
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06708633 91258575
Estimation of prevalence of Helicobacter pylori infection in an asymptomatic elderly population comparing [14C] urea breath test and serology.
Newell DG; Hawtin PR; Stacey AR; MacDougall MH; Ruddle AC
Public Health Laboratory Service Centre for Applied Microbiology and Research, Porton Down, Salisbury, Wiltshire.
Journal of clinical pathology (ENGLAND) May 1991, 44 (5) p385-7, ISSN 0021-9746 Journal Code: HP3
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/94
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

06708144 91257543
A novel enzyme immunoassay for serodiagnosis of Helicobacter pylori **infection.**
Sugiyama T; Imai K; Yoshida H; Takayama Y; Yabana T; Yokota K; Oguma K; Yachi A
Department of Internal Medicine, Sapporo Medical College, Japan.
Gastroenterology (UNITED STATES) Jul 1991, 101 (1) p77-83, ISSN 0016-5085 Journal Code: FH3
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/95
DIALOG(R)File 155:MEDLINE(R)
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06708124 91257508
Positive serum antibody and negative tissue staining for Helicobacter pylori **in subjects with atrophic body gastritis [see comments]**
Karnes WE Jr; Samloff IM; Siurala M; Kekki M; Sipponen P; Kim SW; Walsh JH
Center for Ulcer Research and Education, Veterans Administration Medical Center, Los Angeles, California
Gastroenterology (UNITED STATES) Jul 1991, 101 (1) p167-74, ISSN 0016-5085 Journal Code: FH3
Contract/Grant No.: DK17328, NIDDK
Comment in Gastroenterology 1992 Feb;102(2):744-5
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/96
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

06500744 91041503
Detection of antibodies to Helicobacter pylori **with the immunoenzyme test and indirect immunofluorescence]**
Nachweis von Antikorpern gegen **Helicobacter pylori** mit Enzymimmuntest

und indirekter Immunfluoreszenz.
Abb J; Striegel K; Fruhmorgen P
Mikrobiologisches Institut, Krankenanstalten Ludwigsburg.
Leber, Magen, Darm (GERMANY) Sep 1990, 20 (5) p224-30, ISSN 0300-8622
Journal Code: L3P
Languages: GERMAN Summary Languages: ENGLISH
Document type: JOURNAL ARTICLE ; English Abstract

12/3/97
DIALOG(R)File 155:MEDLINE(R)

06429894 90338484
Comparison of ELISA antigen preparations alone or in combination for serodiagnosing Helicobacter pylori infections.
Hirschl AM; Rathbone BJ; Wyatt JI; Berger J; Rotter ML
Hygiene-Institute, University of Vienna, Austria.
Journal of clinical pathology (ENGLAND) Jun 1990, 43 (6) p511-3,
ISSN 0021-9746 Journal Code: HT3
Languages: ENGLISH
Document type: JOURNAL ARTICLE

12/3/98
DIALOG(R)File 155:MEDLINE(R)

06406478 90282212
Endoscopy in dyspeptic patients: is gastric mucosal biopsy useful? [see comments]
Vaira D; Holton J; Osborn J; D'Anna L; Romanos A; Falzon M; McNeil I
Department of Gastroenterology, Middlesex Hospital, London, England.
American journal of gastroenterology (UNITED STATES) Jun 1990, 85 (6)
p701-4, ISSN 0002-9270 Journal Code: 3HE
Comment in Am J Gastroenterol 1991 May;86(5):647-8
Languages: ENGLISH
Document type: JOURNAL ARTICLE
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 File 155:MEDLINE(R)  1966-2000/Dec W4
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   *File 155: First Medline 2001 update is expected towards the end of
   February. For other NLM information see Help News155.

         Set  Items  Description
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   ?e atpase

   Ref    Items    RT  Index-term
   E1        18       ATPASA
   E2         2       ATPASAS
   E3     46015     1 *ATPASE
   E4      1862       ATPASE //CA(2+) MG(2+) (CA(2+) MG(2+)-ATPASE)
   E5      7693       ATPASE //CA(2+)-TRANSPORTING (CA(2+)-TRANSPORTING ATPASE)
   E6      1140       ATPASE //DYNEIN (DYNEIN ATPASE)
   E7       715       ATPASE    //H(+)-K(+)-EXCHANGING    (H(+)-K(+)-EXCHANGING
                      ATPASE)
   E8      1095       ATPASE //MYOSIN (MYOSIN ATPASE)
   E9     12489       ATPASE   //NA(+)-K(+)-EXCHANGING   (NA(+)-K(+)-EXCHANGING
                      ATPASE)
   E10      180       ATPASE INHIBITORY PROTEIN
   E11        1       ATPASE INHIBITORY PROTEIN, APROTININ
   E12        2       ATPASE P72

            Enter P or PAGE for more
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   Ref    Items    RT  Index-term
   E13        2       ATPASE 6
   E14        1       ATPASE 6/8
   E15        1       ATPASE 8
   E16        2       ATPASE-BINDING PROTEIN (26,500)
   E17        3       ATPASE-DATPASE
   E18        0     1 ATPASE, ACTOMYOSIN
   E19        0     1 ATPASE, CALCIUM
   E20        0     1 ATPASE, CALCIUM MAGNESIUM
   E21        0     1 ATPASE, DYNEIN
   E22        0     1 ATPASE, F0
   E23        0     1 ATPASE, F1
   E24        0     1 ATPASE, HYDROGEN, POTASSIUM

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   ?p

   Ref    Items    RT  Index-term
   E25        0     1 ATPASE, MAGNESIUM
   E26        0     1 ATPASE, MYOSIN
   E27        0     1 ATPASE, SODIUM, POTASSIUM
   E28        1       ATPASEACTIVITY
   E29        1       ATPASEEACTION
   E30       18       ATPASEN
   E31        1       ATPASENACHWEIS
   E32     3198       ATPASES
   E33        4       ATPASE1
   E34        1       ATPASE2
   E35        2       ATPASE2A
   E36        1       ATPASE4

            Enter P or PAGE for more
   ?s e3 or e7 or e32
             46015  ATPASE
               715  ATPASE //H(+)-K(+)-EXCHANGING
              3198  ATPASES
         S1   46862  "ATPASE" OR "ATPASE //H(+)-K(+)-EXCHANGING" OR "ATPASES"
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Ref  Items  Index-term
E37     20  ATPASE6
E38      1  ATPASE7A
E39      1  ATPASE7B
E40     11  ATPASE8
E41      2  ATPASE9
E42     24  ATPASI
E43      6  ATPASIC
E44      3  ATPASICA
E45      2  ATPASICAS
E46      1  ATPASICHE
E47      1  ATPASICO
E48     18  ATPASIQUE

          Enter P or PAGE for more
?e e24

Ref   Items Type  RT  Index-term
R1        0        1 *ATPASE, HYDROGEN, POTASSIUM
R2     1148   X    7  H(+)-K(+)-EXCHANGING ATPASE
?s r1-r2
              0  ATPASE, HYDROGEN, POTASSIUM
           1148  H(+)-K(+)-EXCHANGING ATPASE
      S2   1148  R1-R2
?e r2

Ref   Items Type  RT  Index-term
R1     1148        7 *H(+)-K(+)-EXCHANGING ATPASE
R2      715   X      DC=D8.586.277.40.25.300. (H(+)-K(+)-EXCHANGING ATPAS-
                     E)
R3        0   X    1  ADENOSINETRIPHOSPHATASE, HYDROGEN, POTASSIUM
R4        0   X    1  ATPASE, HYDROGEN, POTASSIUM
R5        0   X    1  H(+)-K(+)-TRANSPORTING ATPASE
R6        0   X    1  HYDROGEN, POTASSIUM ATPASE
R7        0   X    1  HYDROGEN, POTASSIUM, ADENOSINETRIPHOSPHATASE
R8    26771   B   11  ADENOSINETRIPHOSPHATASE
?s r1-r8
           1148  H(+)-K(+)-EXCHANGING ATPASE
            715  DC=D8.586.277.40.25.300.
              0  ADENOSINETRIPHOSPHATASE, HYDROGEN, POTASSIUM
              0  ATPASE, HYDROGEN, POTASSIUM
              0  H(+)-K(+)-TRANSPORTING ATPASE
              0  HYDROGEN, POTASSIUM ATPASE
              0  HYDROGEN, POTASSIUM, ADENOSINETRIPHOSPHATASE
          26771  ADENOSINETRIPHOSPHATASE
      S3  27423  R1-R8
?e r8

Ref   Items Type  RT  Index-term
R1    26771       11 *ADENOSINETRIPHOSPHATASE
R2    26056   X      DC=D8.586.277.40.25. (ADENOSINETRIPHOSPHATASE)
R3    46015   X    1  ATPASE
R4      316   B   49  ACID ANHYDRIDE HYDROLASES
R5     2835   N   11  CA(2+) MG(2+)-ATPASE
R6     7693   N   11  CA(2+)-TRANSPORTING ATPASE
R7     1140   N    9  DYNEIN ATPASE
R8     1148   N    7  H(+)-K(+)-EXCHANGING ATPASE
R9     5071   N   12  H(+)-TRANSPORTING ATP SYNTHASE
R10    1248   N    5  KINESIN
R11    1095   N   11  MYOSIN ATPASE
R12   12489   N   14  NA(+)-K(+)-EXCHANGING ATPASE
?s r1-r3
          26771  ADENOSINETRIPHOSPHATASE
          26056  DC=D8.586.277.40.25.
          46015  ATPASE
      S4  59018  R1-R3
?ds
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Set     Items   Description
S1      46862   "ATPASE" OR "ATPASE //H(+)-K(+)-EXCHANGING" OR "ATPASES"
S2       1148   R1-R2
S3      27423   R1-R8
S4      59018   R1-R3
?s (s1-s5) and (helicobact? or pylori or pylor or pyloris or pyloridis or hpylori or hf
elis)
>>>"S5" does not exist
>>>Both terms in the range must be of the same type
?s (s1-s4) and (helicobact? or pylori or pylor or pyloris or pyloridis or hpylori or hf
elis)
           46862  S1
            1148  S2
           27423  S3
           59018  S4
           11767  HELICOBACT?
           12258  PYLORI
               4  PYLOR
               9  PYLORIS
             175  PYLORIDIS
               0  HPYLORI
               0  HFELIS
      S5     133  (S1-S4) AND (HELICOBACT? OR PYLORI OR PYLOR OR PYLORIS OR
                  PYLORIDIS OR HPYLORI OR HFELIS)
?e pepsinogen

Ref    Items    RT  Index-term
E1         4        PEPSINOBRAZUIUSHCHAIA
E2         1        PEPSINOBRAZUIUSHCHEI
E3      1838     1 *PEPSINOGEN
E4        11        PEPSINOGEN (1-12)
E5       126     7  PEPSINOGEN A
E6         9        PEPSINOGEN A --ANALYSIS --AN
E7         1        PEPSINOGEN A --ANTAGONISTS AND INHIBITORS --AI
E8         2        PEPSINOGEN A --BIOSYNTHESIS --BI
E9        56        PEPSINOGEN A --BLOOD --BL
E10       12        PEPSINOGEN A --CHEMISTRY --CH
E11        3        PEPSINOGEN A --DRUG EFFECTS --DE
E12       10        PEPSINOGEN A --GENETICS --GE

          Enter P or PAGE for more
?p

Ref    Items    RT  Index-term
E13        2        PEPSINOGEN A --IMMUNOLOGY --IM
E14        2        PEPSINOGEN A --ISOLATION AND PURIFICATION --IP
E15       13        PEPSINOGEN A --METABOLISM --ME
E16       10        PEPSINOGEN A --SECRETION --SE
E17        1        PEPSINOGEN A --URINE --UR
E18        0     1  PEPSINOGEN B
E19       23     4  PEPSINOGEN C
E20        1        PEPSINOGEN C --ANALYSIS --AN
E21        1        PEPSINOGEN C --ANTAGONISTS AND INHIBITORS --AI
E22        1        PEPSINOGEN C --BIOSYNTHESIS --BI
E23       13        PEPSINOGEN C --BLOOD --BL
E24        4        PEPSINOGEN C --CHEMISTRY --CH

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?s e5-e17
             126  PEPSINOGEN A
               9  PEPSINOGEN A --ANALYSIS --AN
               1  PEPSINOGEN A --ANTAGONISTS AND INHIBITORS --AI
               2  PEPSINOGEN A --BIOSYNTHESIS --BI
              56  PEPSINOGEN A --BLOOD --BL
              12  PEPSINOGEN A --CHEMISTRY --CH
               3  PEPSINOGEN A --DRUG EFFECTS --DE
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              10  PEPSINOGEN A --GENETICS --GE
               2  PEPSINOGEN A --IMMUNOLOGY --IM
               2  PEPSINOGEN A --ISOLATION AND PURIFICATION --IP
              13  PEPSINOGEN A --METABOLISM --ME
              10  PEPSINOGEN A --SECRETION --SE
               1  PEPSINOGEN A --URINE --UR
      S6     126  E5-E17
?e e5

Ref    Items Type  RT  Index-term
R1       126        7 *PEPSINOGEN A
R2        98   X       DC=D8.444.509.700. (PEPSINOGEN A)
R3      1838   X    1  PEPSINOGEN
R4         0   X    1  PEPSINOGEN I
R5         0   X    1  PEPSINOGEN 3, GROUP I
R6         0   X    1  PEPSINOGEN 5, GROUP I
R7        49   X    1  UROPEPSINOGEN
R8      1745   B    6  PEPSINOGENS
?s r1-r7
             126  PEPSINOGEN A
              98  DC=D8.444.509.700.
            1838  PEPSINOGEN
               0  PEPSINOGEN I
               0  PEPSINOGEN 3, GROUP I
               0  PEPSINOGEN 5, GROUP I
              49  UROPEPSINOGEN
      S7    1872  R1-R7
?e r8

Ref    Items Type  RT  Index-term
R1      1745        6 *PEPSINOGENS
R2      1690   X       DC=D8.444.509. (PEPSINOGENS)
R3         0   X    1  PEPSINOGEN B
R4     13711   R    6  GASTRIC JUICE
R5      6119   B   13  ENZYME PRECURSORS
R6       126   N    7  PEPSINOGEN A
R7        23   N    4  PEPSINOGEN C
?e pga

Ref    Items   RT  Index-term
E1         8       PG-M CORE PROTEIN
E2         1       PG-1
E3       909    1 *PGA
E4         0    1  PGA SYNTHETIC
E5        11       PGAA
E6         1       PGAA PROTEIN
E7         1       PGAAAG
E8         1       PGAATTC
E9         4       PGAB
E10        3       PGABA
E11        1       PGABAS
E12        1       PGABX2

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?s e3
      S8     909  "PGA"
?e pg1

Ref    Items  Index-term
E1         1  PG0400
E2        13  PG05
E3        95 *PG1
E4         1  PG1*2
E5         1  PG1A
E6         1  PG1ALPHA
E7         1  PG1NA
E8         2  PG10
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E9        1  PG10.2 PROTEIN
E10       1  PG100
E11       1  PG101
E12      10  PG11

          Enter P or PAGE for more
?s e3
      S9      95  "PG1"
?ds

Set     Items   Description
S1      46862   "ATPASE" OR "ATPASE //H(+)-K(+)-EXCHANGING" OR "ATPASES"
S2       1148   R1-R2
S3      27423   R1-R8
S4      59018   R1-R3
S5        133   (S1-S4) AND (HELICOBACT? OR PYLORI OR PYLOR OR PYLORIS OR -
                 PYLORIDIS OR HPYLORI OR HFELIS)
S6        126   E5-E17
S7       1872   R1-R7
S8        909   "PGA"
S9         95   "PG1"
?s s5 and (s6-s9)
             133  S5
             126  S6
            1872  S7
             909  S8
              95  S9
     S10       4  S5 AND (S6-S9)
?t s10/9/all

 10/9/1
DIALOG(R)File 155:MEDLINE(R)
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09774848   99070725
   Serum antibodies to H+,K+- ATPase , serum pepsinogen A and
 Helicobacter pylori in relation to gastric mucosa morphology in
patients with low or low-normal concentrations of serum cobalamins.
  Lindgren A; Burman P; Kilander AF; Nilsson O; Lindstedt G
  Department of Internal Medicine, Sahlgrenska University Hospital,
Goteborg, Sweden.
  European journal of gastroenterology & hepatology (ENGLAND) Jul 1998,
10 (7) p583-8, ISSN 0954-691X Journal Code: B9X
  Languages: ENGLISH
  Document type: JOURNAL ARTICLE
  JOURNAL ANNOUNCEMENT: 9904
  Subfile:  INDEX MEDICUS
  OBJECTIVES: To compare the diagnostic performance of serum antibodies to
H+,K+- ATPase (EC 3.6.1.36), serum pepsinogen A (EC 3.4.23.1) and the
Schilling test in diagnosing chronic atrophic body gastritis; to study the
interrelationships between H+,K+- ATPase antibodies, serology for
 Helicobacter pylori , and gastric morphology. DESIGN: Patients with
suspected cobalamin deficiency and serum cobalamin < 200 micromol/l were
investigated using upper gastrointestinal endoscopy, the Schilling test and
serum tests for H+,K+-ATPase antibodies, pepsinogen A, and H. pylori .
SETTING: The Department of Internal Medicine, Sahlgrenska University
Hospital, Goteborg, Sweden. PATIENTS: Ninety seven consecutively referred
patients. MAIN OUTCOME MEASURES: Sensitivity and specificity of assays for
serum H+,K+- ATPase antibodies, serum pepsinogen A, and the Schilling
test. RESULTS: Assays of serum antibodies to H+,K+-ATPase and of serum
 pepsinogen A displayed equal diagnostic sensitivity for atrophic
gastritis (around 0.90 for the severe forms) and higher than that for the
Schilling test (0.65). The diagnostic specificity for pepsinogen A (1.0)
was higher than for H+,K+-ATPase antibodies (about 0.80). The prevalence
of antral gastritis and positivity for H. pylori antibodies declined with
the transition of body gastritis into severe atrophy, while the prevalence
of H+,K+- ATPase antibodies increased. CONCLUSION: Pepsinogen A is
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preferable to serum H+,K+-**ATPase** antibodies in the diagnosis of gastric body mucosal atrophy. The formation of H+,K+-**ATPase** antibodies does not seem to be a primary event in the development of gastric body muscosal atrophy.

Tags: Human

Descriptors: Antibodies, Bacterial--Blood--BL; *Gastritis, Atrophic --Diagnosis--DI; * **H(+)-K(+)-Exchanging ATPase --Bloo** d--BL; * **Helicobacter pylori** --Immunology--IM; * **Pepsinogen** A--Blood--BL; *Vitamin B 12--Blood--BL; Adult; Aged; Chronic Disease; Gastric Mucosa --Immunology--IM; Gastric Mucosa--Pathology--PA; Gastritis, Atrophic --Immunology--IM; **H(+)-K(+)-Exchanging ATPase --Immu** nology--IM; Middle Age; **Pepsinogen** A--Immunology--IM; Reference Values; Schilling Test; Sensitivity and Specificity; Serologic Tests; Vitamin B 12--Immunology--IM

CAS Registry No.: 0 (Antibodies, Bacterial); 68-19-9 (Vitamin B 12); 9001-10-9 (Pepsinogen A)

Enzyme No.: EC 3.6.1.36 (H(+)-K(+)-Exchanging **ATPase**)

10/9/2

DIALOG(R)File 155:MEDLINE(R)

07425784 92221233

Helicobacter pylori **and hypergastrinaemia during proton pump inhibitor therapy.**

McColl KE; Nujumi AM; Dorrian CA; Macdonald AM; Fullarton GM; Harwood J

University Dept. of Medicine and Therapeutics, Western Infirmary, Glasgow, Scotland.

Scandinavian journal of gastroenterology (NORWAY) 1992, 27 (2) p93-8, ISSN 0036-5521 Journal Code: UCS

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9207

Subfile: INDEX MEDICUS

The rise in serum gastrin and **pepsinogen** I after 5 days' treatment with the proton pump inhibitor pantoprazole (40 mg/day) was examined in eight duodenal ulcer patients with **Helicobacter pylori** infection and compared with eight in whom it had been eradicated. Before treatment, the post-prandial serum gastrin concentrations were higher in the H. **pylori** -positive than -eradicated patients (p less than 0.05). The median rise in pre-prandial serum gastrin concentrations on treatment was similar in the H. **pylori** -positive (41%) and -eradicated patients (45%). The rise in post-prandial serum gastrin was also similar in the H. **pylori** -positive (81%) and -eradicated patients (69%), resulting in significantly higher gastrin concentrations during treatment in the former. The median rise in serum **pepsinogen** I on treatment was greater in the H. **pylori** -positive (114%) than in the -eradicated patients (8%), resulting in significantly higher concentrations during treatment in the former. These observations indicate that eradication of H. **pylori** may be a means of moderating the hypergastrinaemia caused by acid-inhibitory therapy. They also indicate that H. **pylori** -related hypergastrinaemia is not due to an increase of the antral surface pH by the bacterium's urease activity.

Tags: Human; Male; Support, Non-U.S. Gov't

Descriptors: **Adenosinetriphosphatase** --Antagonists and Inhibitors--AI; *Benzimidazoles--Pharmacology--PD; *Gastrins--Blood--BL; * **Helicobacter pylori** ; ***Helicobacter** Infections--Blood--BL; *Pepsinogens--Blood--BL; *Peptide Fragments--Blood--BL; *Sulfoxides--Pharmacology--PD; Benzimidazole s--Therapeutic Use--TU; Duodenal Ulcer--Blood--BL; Duodenal Ulcer--Drug Therapy--DT; Duodenal Ulcer--Metabolism--ME; Gastric Acidity Determination ; Gastrins--Drug Effects--DE; **Helicobacter** Infections--Metabolism--ME; Pepsinogens--Drug Effects--DE; Peptide Fragments--Drug Effects--DE; Sulfoxides--Therapeutic Use--TU

CAS Registry No.: 0 (Benzimidazoles); 0 (Gastrins); 0 (Pepsinogens) ; 0 (Peptide Fragments); 0 (Sulfoxides); 102625-70-7 (pantoprazole); 75903-15-0 (pepsinogen (1-12))

Enzyme No.: EC 3.6.1.3 **Adenosinetriphosphatase**)

10/9/3
DIALOG(R)File 155:MEDLINE(R)

06651765 91047803
Acid and barriers. Current research and future developments for peptic ulcer therapy.
Rademaker JW; Hunt RH
Division of Gastroenterology, McMaster University Medical Centre, Hamilton, Ontario, Canada.
Scandinavian journal of gastroenterology. Supplement (NORWAY) 1990, 175 p19-26, ISSN 0085-5928 Journal Code: UCT
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
JOURNAL ANNOUNCEMENT: 9102
Subfile: INDEX MEDICUS
Medical therapy for peptic ulcer disease has been targeted at inhibiting acid secretion based on the belief that ulcers occur due to an imbalance between aggressive and protective factors. New antisecretory agents are unlikely to show any dramatic improvement over the success and safety of histamine H2 receptor antagonists or the recently introduced H+K+**ATPase** proton pump antagonist omeprazole. The development of specific muscarinic M3 and gastrin receptor antagonists will provide useful agents to suppress acid and **pepsinogen** secretion by alternative means and may prevent the associated hypergastrinaemia seen with anti-secretory therapy. Enhancement of mucosal defence by site protective agents will be based on a better understanding of the vascular and immune factors involved in maintaining mucosal integrity and the growth factors that regulate wound healing. Molecular techniques are likely to produce the 'model anti-ulcer' agent which will effectively inhibit acid secretion and also enhance wound healing thus providing a cure for this chronic disease. (67 Refs.)
Tags: Human
Descriptors: *Antacids--Therapeutic Use--TU; *Anti-Ulcer Agents --Therapeutic Use--TU; *Peptic Ulcer--Drug Therapy--DT; Gastric Mucosa --Physiology--PH; **Helicobacter pylori** ; **Helicobacter** Infections --Complications--CO; Histamine H2 Antagonists--Therapeutic Use--TU; Intestinal Mucosa--Physiology--PH; Peptic Ulcer--Etiology--ET; Wound Healing--Physiology--PH
CAS Registry No.: 0 (Antacids); 0 (Anti-Ulcer Agents); 0 (Histamine H2 Antagonists)

10/9/4
DIALOG(R)File 155:MEDLINE(R)

06279395 89283673
NSAIDs: new approaches to limiting gastropathy.
Zeidler H; Munzel P
Abt. Rheumatologie, Zentrum Innere Medizin und Dermatologie, Medizinische Hochschule, Hanover, West Germany.
Scandinavian journal of rheumatology. Supplement (SWEDEN) 1989, 78 p18-23; discussion 30-2, ISSN 0301-3847 Journal Code: UDO
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
JOURNAL ANNOUNCEMENT: 8909
Subfile: INDEX MEDICUS
An extensive literature search on non-steroidal anti-inflammatory drug (NSAID)-induced gastropathy in rheumatic conditions has been carried out. A reduced incidence of gastropathy has been observed among newly developed NSAIDs such as etodolac and the non-acidic nabumetone. An alternative prophylactic therapy to avoid NSAID-induced gastroduodenal mucosal damage which has been successfully tested in several trials is co-medication with the prostaglandin analogue misoprostol. The cytoprotective agent sucralfate also appears to be effective. Recent observations of Campylobacter **pylori** infections in NSAID-induced gastropathy introduces the question as to

whether simultaneous antibacterial medication should be routinely administered during NSAID therapy. At present the invasive technique of endoscopy is used to ascertain gastroduodenal mucosal damage. However, a new technique which merely requires blood sampling is being investigated. This involves measurement of serum levels of the precursor molecules for the gastric enzyme pepsin, **pepsinogen** I and II. In future this assay could constitute a non-invasive method for detecting gastroduodenal mucosal damage. (9 Refs.)

Tags: Human

Descriptors: *Anti-Inflammatory Agents, Non-Steroidal--Adverse Effects --AE; *Stomach Diseases--Prevention and Control--PC; **Adenosinetriphosphatase** --Antagonists and Inhibitors--AI; Anti-Inflammatory Agents, Non-Steroidal--Therapeutic Use--TU; Campylobacter Infections--Complications --CO; Campylobacter Infections--Drug Therapy--DT; Chemistry, Pharmaceutical; Drug Therapy, Combination; Pepsinogens--Blood--BL; Prostaglandins--Therapeutic Use--TU; Risk Factors; Stomach Diseases --Chemically Induced--CI; Stomach Diseases--Complications--CO; Sucralfate --Therapeutic Use--TU

CAS Registry No.: 0 (Pepsinogens); 0 (Prostaglandins); 54182-58-0 (Sucralfate)

Enzyme No.: EC 3.6.1.3 **Adenosinetriphosphatase**)

?logoff hold

23feb01 10:33:36 User228206 Session D1422.2

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$0.80 4 Type(s) in Format 9

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$6.11 Estimated cost File155

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$6.46 Estimated cost this search

$6.47 Estimated total session cost 1.835 DialUnits

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Helicobacter pylori **and hypergastrinaemia during proton pump inhibitor therapy.**

McColl KE; Nujumi AM; Dorrian CA; Macdonald AM; Fullarton GM; Harwood J
University Dept. of Medicine and Therapeutics, Western Infirmary, Glasgow, Scotland.

Scandinavian journal of gastroenterology (NORWAY) 1992, 27 (2) p93-8, ISSN 0036-5521 Journal Code: UCS
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9207
Subfile: INDEX MEDICUS

The rise in serum gastrin and **pepsinogen** I after 5 days' treatment with the proton pump inhibitor pantoprazole (40 mg/day) was examined in eight duodenal ulcer patients with **Helicobacter pylori** infection and compared with eight in whom it had been eradicated. Before treatment, the post-prandial serum gastrin concentrations were higher in the H. **pylori** -positive than -eradicated patients (p less than 0.05). The median rise in pre-prandial serum gastrin concentrations on treatment was similar in the H. **pylori** -positive (41%) and -eradicated patients (45%). The rise in post-prandial serum gastrin was also similar in the H. **pylori** -positive (81%) and -eradicated patients (69%), resulting in significantly higher gastrin concentrations during treatment in the former. The median rise in serum **pepsinogen** I on treatment was greater in the H. **pylori** -positive (114%) than in the -eradicated patients (8%), resulting in significantly higher concentrations during treatment in the former. These observations indicate that eradication of H. **pylori** may be a means of moderating the hypergastrinaemia caused by acid-inhibitory therapy. They also indicate that H. **pylori** -related hypergastrinaemia is not due to an increase of the antral surface pH by the bacterium's urease activity.

Tags: Human; Male; Support, Non-U.S. Gov't

NSAIDs: new approaches to limiting gastropathy.

Zeidler H; Munzel P

Abt. Rheumatologie, Zentrum Innere Medizin und Dermatologie, Medizinische Hochschule, Hanover, West Germany.

Scandinavian journal of rheumatology. Supplement (SWEDEN) 1989, 78 p18-23; discussion 30-2, ISSN 0301-3847 Journal Code: UD0

Languages: ENGLISH

Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

JOURNAL ANNOUNCEMENT: 8909

Subfile: INDEX MEDICUS

An extensive literature search on non-steroidal anti-inflammatory drug (NSAID)-induced gastropathy in rheumatic conditions has been carried out. A reduced incidence of gastropathy has been observed among newly developed NSAIDs such as etodolac and the non-acidic nabumetone. An alternative prophylactic therapy to avoid NSAID-induced gastroduodenal mucosal damage which has been successfully tested in several trials is co-medication with the prostaglandin analogue misoprostol. The cytoprotective agent sucralfate also appears to be effective. Recent observations of Campylobacter **pylori** infections in NSAID-induced gastropathy introduces the question as to whether simultaneous antibacterial medication should be routinely administered during NSAID therapy. At present the invasive technique of endoscopy is used to ascertain gastroduodenal mucosal damage. However, a new technique which merely requires blood sampling is being investigated. This involves measurement of serum levels of the precursor molecules for the gastric enzyme pepsin, **pepsinogen** I and II. In future this assay could constitute a non-invasive method for detecting gastroduodenal mucosal damage. (9 Refs.)

Tags: Human

Acid and barriers. Current research and future developments for peptic ulcer therapy.

Rademaker JW; Hunt RH

Division of Gastroenterology, McMaster University Medical Centre, Hamilton, Ontario, Canada.

Scandinavian journal of gastroenterology. Supplement (NORWAY) 1990, 175 p19-26, ISSN 0085-5928 Journal Code: UCT

Languages: ENGLISH

Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

JOURNAL ANNOUNCEMENT: 9102

Subfile: INDEX MEDICUS

Medical therapy for peptic ulcer disease has been targeted at inhibiting acid secretion based on the belief that ulcers occur due to an imbalance between aggressive and protective factors. New antisecretory agents are unlikely to show any dramatic improvement over the success and safety of histamine H2 receptor antagonists or the recently introduced H+K+**ATPase** proton pump antagonist omeprazole. The development of specific muscarinic M3 and gastrin receptor antagonists will provide useful agents to suppress acid and **pepsinogen** secretion by alternative means and may prevent the associated hypergastrinaemia seen with anti-secretory therapy. Enhancement of mucosal defence by site protective agents will be based on a better understanding of the vascular and immune factors involved in maintaining mucosal integrity and the growth factors that regulate wound healing. Molecular techniques are likely to produce the 'model anti-ulcer' agent which will effectively inhibit acid secretion and also enhance wound healing thus providing a cure for this chronic disease. (67 Refs.)

Tags: Human